

In Capsule Series

Internal Medicine

NEUROLOGY

by

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Hemiplegia

Definition :

Paralysis of one side of the body due to pyramidal tract lesion (UMNL) anywhere from its origin in the cerebral cortex down to the 5th cervical segment of the spinal cord (C5).

Etiology :

1- **Vascular (Stroke)** : the **commonest cause** of hemiplegia.

I. **Ischemic :** (Cerebral infarction) *85% of all strokes.*

a) **Thrombosis :**

➡ Atherosclerosis : The most common cause.

✓ Extracranial : aorta , carotid or vertebral

✓ Intracranial : ACA , MCA , PICA

➡ Arteritis, Dissection.

➡ Hematological disorders : Polycythemia, sickle cell anemia & hypercoagulability state.

b) **Embolism :** either from heart or carotid

➡ From the heart :

✓ Atrial fibrillation (AF)

✓ Atrial myxoma

✓ Infective endocarditis.

✓ Dilated cardiomyopathy

✓ Mitral Stenosis

✓ Congenital heart disease.

✓ Myocardial infarction.

➡ From carotid vessels.

Risk factors for ischemic stroke

- Risk factors of atherosclerosis : modifiable & non modifiable ... see cardiology
- Transient ischemic attacks.

II. **Hemorrhagic :** *accounts for 15% of all strokes*

a. **Intracerebral :** 2 HAT

- Hemorrhagic blood diseases: purpura
- Hypertension
- Aneurism
- Angiomatous malformation
- Trauma
- Tumor

b. **Subarachnoid hemorrhage :** 2A , 2H , 2T see later

2-Congenital : cerebral palsy

3- Trauma.

4-Inflammatory : meningitis , encephalitis.

5-Neoplastic : meningioma.

6-Demyelinating : multiple sclerosis.

7- Hysterical.

Acute onset of hemiplegia :
Vascular, infectious & trauma.

Stages of hemiplegia :

1) Shock stage (flaccid paralysis) :

- ✎ Occur only in cases with acute onset (as embolism & trauma).
- ✎ Initially, the paralysis may show the criteria of **LMNL** (hypotonia, hyporeflexia) but the planter response is extensor.
- ✎ Increased tone (spasticity) develops after hours, days or rarely weeks.
- ✎ The more the prolongation of this stage, the worse the prognosis.

2) Stage of spastic paralysis :

- ✎ It is the stage of established Hemiplegia.
- ✎ The paralyzed muscles show the criteria of **UMNL** (hypertonia, hyperreflexia)

Pyramidal lesion distribution :

- It affects the Distal more than proximal muscles. So, skilled movements of fingers & toes are more affected.
- The antigravity muscles are more spastic than progravity muscles. So, progravity muscles are weaker than antigravity.
 - U.L → Flexors are more spastic & Extensors are weaker.
 - L.L → Extensors are more spastic & Flexors are weaker.
 - In both limbs the Adductors are more spastic than Abductors.
- The spastic muscles show “clasp-knife” phenomenon : strong resistance followed by sudden relaxation of the muscles.

Levels of Hemiplegia :

44623

I. Cortical hemiplegia :

4



- 1) It starts usually as contralateral monoplegia not hemiplegia. (due to wide distributions of Betz cells in area 4)
- 2) History of Convulsions before the onset of paralysis (irritation of area 4)
- 3) **Coma** if lesion is extensive.
- 4) Other cortical manifestations :
 - Contralateral cortical sensory loss.
 - Aphasia.
 - Agraphia.....

II. Subcortical hemiplegia :

4



- ✓ The same 4 criteria as cortical but the paralysis is **more extensive**.
- ✓ **Coma and convulsions** are less extensive.

III. Capsular hemiplegia :

6



- 1) **Complete hemiplegia** from the start on the opposite side of the lesion (contralateral)
- 2) **Hemianesthesia** in the opposite side of the lesion (same side of the paralysis)
- 3) **Homonymous hemianopia.**
- 4) **3 No** : No Coma, No Convulsions, No other Cortical manifestations.
- 5) Paralysis of the muscles of the lower part of the face **on the opposite side of the lesion** (the Same side of hemiplegia)
- 6) Hypoglossal paralysis on the opposite site of the lesion : Deviation of the tongue to the side of paralysis.

All motor muscles receive bilateral pyramidal supply except the lower half of 7th & 12th.

IV. Brain stem hemiplegia(Crossed hemiplegia) :

2



- 1) Hemiplegia is on the opposite side of the lesion.
- 2) Motor cranial nerve affection of the same side of the lesion due to affection of the cranial nuclei in the Brain stem (LMNL)

The cranial nerve affection differs according to the site of the lesion in the brainstem

➡ **Mid brain lesion :**

- Weber's syndrome : Ipsilateral 3rd cranial nerve paralysis & contralateral hemiplegia
- Benedict's syndrome : like Weber plus contralateral ataxia due to affection of the red nucleus.

➡ **Pons lesion :**

- Millard Gubler syndrome : Ipsilateral 6th & 7th cranial nerve paralysis & contralateral hemiplegia.

- Foville syndrome : Loss of conjugate movement of the eyes due to lesion in the medial longitudinal bundle (MLB) & contralateral hemiplegia.

➔ Medullary lesion :

- Avellis syndrome : Ipsilateral 9th & 10th cranial nerve paralysis (palate-pharyngeal-laryngeal paralysis) & contralateral hemiplegia.

V. Spinal hemiplegia : (*Brown Sequard syndrome*) **3**



- 1) Hemiplegia on the same side of lesion.
- 2) Contralateral loss of pain & temperature sensation.
- 3) Touch sensation is often clinically normal.

Complications :

1. Pneumonia.
2. DVT & Pulmonary embolism.
3. Psychosis.
4. Bed sores.
5. Constipation.
6. Retention of urine (psychic).
7. Epilepsy.
8. Frozen shoulder.
9. Wasting of the muscles (Disuse atrophy).

Investigations :

1- First line investigations :

- CBC & ESR.
- INR, PTT
- Plasma glucose, cholesterol, urea, electrolytes.
- Urinalysis.
- ECG.

2- Cerebral imaging :

i. CT brain :

- To exclude non vascular causes e.g. tumor.
- To differentiate an ischemic stroke (hypodense blackish lesion) from hemorrhagic stroke (hyperdense whitish lesion)

ii. **MRI** : Usually detects area of evolving infarction within hours, CT is sometimes negative for up to several days after acute infarction.

3- Lumbar puncture : in hemorrhagic stroke.

4- Echocardiography : if cardiogenic embolism is suspected.

5- Duplex carotid ultrasound : When carotid artery occlusion is suspected.

6- Angiography : it plays an important role in the preoperative evaluation of carotid artery disease.

Leading causes of death :

Causes of death after stroke is not related primarily to the stroke itself.

- Pneumonia.
- Pulmonary embolism.
- Ischemic heart disease.

Value of examination of the cranial nerves

- If there is no cranial involvement: the lesion is **spinal hemiplegia**.
- If the only cranial nerve involved is the lower part of 7th&12th: the lesion is usually **capsular hemiplegia**.
- If the cranial nerve is LMNL & affected the opposite side of hemiplegia: the lesion is **brainstem hemiplegia**.

Treatment:

1-General Care of the comatosed patient :

- **Bed:** frequent change in the position to prevent bedsores.
- **Balanced nutrition :** tube feeding.
- **Bladder :** urinary catheter.
- **Bowel :** daily enema.
- **Breathing :** (care of respiration)
 - O2 inhalation.
 - Suction of secretions.
 - antibiotics to prevent chest infection (pneumonia)

2-Sympatomatic treatment :

- Cerebral dehydrating agents: (Manitol + Lasix) to minimize edema.
- Antiemetics & K in severe vomiting.
- Vitamins & tonics.
- Sedative, tranquilizers.
- Muscle relaxants for spasticity.

3-Physiotherapy & speech therapy :

- Regular passive exercise of the paralyzed limbs.
- Positioning of the paralyzed limbs in the optimal position.

4-Control of blood pressure :

- **Ischemic stroke :** Antihypertensive therapy is indicated in severe hypertension ($\geq 220/120$) or in those with hypertensive encephalopathy, aortic dissection, acute pulmonary edema or myocardial infarction.
- **Hemorrhagic stroke :** Antihypertensive therapy is initiated when the systolic blood pressure is > 180 mmHg or the diastolic blood pressure > 105 mmHg.

5-Specific treatment (treatment of the cause)

A-Thrombolytic therapy :

- Indications of thrombolytic therapy in stroke :

- Acute ischemic stroke with clearly definable time of onset.
- 3 hours time window. (time is brain, Nooooo time is a lot of brain !!)
- A baseline CT excluding ICH & SAH.
- Age is 18 years or older.
- Dose of i.v. alteplase (tissue plasminogen activator) : 0.9 mg/kg (max. 90 mg) infused over 1 hour.

- Absolute contraindications : 11

- CT scan today shows intracranial bleeding.
- Symptoms suggestive of subarachnoid hge, even if the CT is normal.
- CT scan today shows multilobar infarction (hypodense area > 1/3 of cerebral hemisphere)
- Prior history of intracranial bleeding.
- Any of the following within the past 3 months : intracranial or intraspinal surgery, serious head trauma.
- Blood pressure > 185 mmHg (systolic) or > 110 mmHg (diastolic)
- Evidence of active internal bleeding.
- Patient has an arteriovenous malformation or aneurysm.
- Lab evidence of coagulopathy : e.g. platelets count < 100000
- Patient received heparin in the past 48 h and PTT above normal range
- Patient received warfarin & INR > 1.7

- Relative contraindications : 4

- Major surgery or serious trauma in past 2 weeks.
- GIT or urinary tract bleeding within past 3 weeks.
- Acute MI in past 3 months or post MI pericarditis.
- Blood glucose < 50 or > 400 mg/dl

B-Anticoagulants : ?

- Indicated only in cardioembolic cases & should be delayed for at least 5 days after an acute event.
- I can say that the only role of anticoagulant therapy following an acute stroke is for prevention of DVT & no one can blame me.
- Method :
 - ✎ Heparin IV : 5000 – 10000 units followed by 1000 u/h or LMWH.
 - ✎ Oral anticoagulants (*marevan*): because of the delayed onset of action, it is started 2-3 days before discontinuation of heparin & is continued for at least 6 months.

C- Anti-platelets :

- Aspirin 300 mg for 2 weeks then 75 mg/d is highly recommended for all cases of ischemic stroke. Clopidogrel 75 mg/d may be used.
- Aspirin reduces the recurrence & mortality rate.

D-cerebral hemorrhage:

- Evacuation of blood surgically provided that it is not deep hemorrhage.
- Anti fibrinolysis : Epsilon amino caproic acid.

6- Neuroprotective not effective

- ✎ Cerebrolysin : nerve growth factor.
- ✎ Trental, Nootropil : decrease O₂ consumption & blood viscosity.

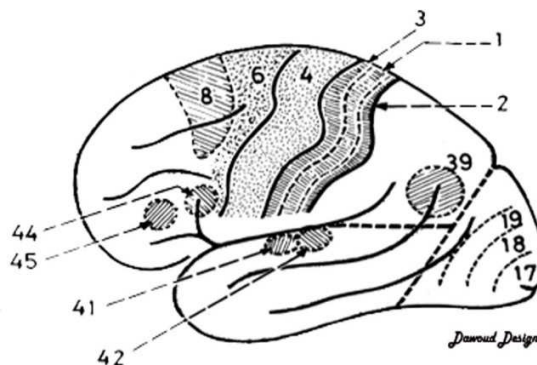
Areas of cerebral cortex

Frontal lobe:

- Area 4: in front of the central sulcus.
Upside down presentation.
- Area 6: posterior to the precentral sulcus.
- Area 8: in the middle frontal gyrus.
- Area 44,45: in the dominant hemisphere.
- Prefrontal area: in the most anterior part.

Aphasia: patient can't express his ideas in spoken words.

Agraphia: patient can't express his ideas in written words.



Parietal lobe:

- Area 1,2,3: posterior to the central sulcus.
Upside down presentation. →

Cortical Sensations

- Midzone temperature.
- Position,Movement.
- Fine touch(tactile localization , tactile discrimination ,stereognosis)

- Area 37: in front of the angular sulcus In the dominant hemisphere.

Apraxia: No practice.

- Area 39: Posterior to the angular sulcus in the dominant hemisphere.

Alaxia: can't read.

Temporal lobe:

- Area 41,42: in the supratemporal gyrus.
- Area 22: adjacent to area 41,42.
- Ancus & hippocampus: smell & memory.

Limbic system → Uncus & hippocampus

Site: in the medial inferior part of the temporal lobe.

Function: smell & memory.

Leison: - olfactory hallucination

- Loss of memory for the last events

Occipital lobe:

- Area 17: reception of visual images.
- Area 18,19: anterior to area 17.
Recognition of the images.

Area 6

Function

partly supplies $\left\{ \begin{array}{l} \text{Pyramidal T.} \\ \text{Extra A.T.} \end{array} \right.$

Lesion:

- Contralateral
- Hypertonia
- Hyperreflexia

Area 4

Function:

initiation, Voluntary motor activity opposite 1/2 of body.

Lesion: irritative

(Contralateral Motor J. Fits)

Site: Convulsions $\rightarrow$ ms $\rightarrow$ one side

onset: Focal onset start $\rightarrow$ Thumb or Angle of H

Spread: Special march. $\rightarrow$ Big Toe.

offset $\rightarrow$ Transient paralysis

Destructive

Contralateral motor paralysis.

Area 1, 2, 3:

Function: perception of Cortical Sensation from opposite 1/2 of body

Lesion:

Irritative/contralateral Sensory Jacksonian Fits
Sensory Fits (Numbness), one side
Focal onset
Special march.

Destructive: contralateral Sensory loss.

Area 8:

Function: Voluntary Conjugate eye deviation To opposite side

Lesion:

Irritative: attack of Conj. eye deviation to opp. side of lesion.

Destructive: loss of To opp. side of lesion.

Area 44

Function

Motor centre for Speech
Responsible for formulation of Spoken words
present in left Cerebral cortex in Rt handed person, vice versa

Lesion

Motor aphasia
(Verbal Aphasia)

Area 45

(writing aphasia)
(Agraphia)

Area 41, 42

Function centre for Hearing.

Lesion

Irritative
Auditory hallucinations.

Destructive
 $\downarrow$ Hearing.
Never complete deafness.

Area 22

Function Recognition and Recall of Sound and heard words.

Lesion Auditory Agnosia.

Can hear, but can't recognize them.

Area 39

Cortical.

In Dominant hemisphere only
Responsible for Storage + recall of ideas of speech + ideas of complex voluntary motor activity.

Reading (Recognition, Recall of letters)

• Apraxia
• Jargon's Aphasia
• Alexia (visual Aphasia)

Area 37

In Dominant hemisphere only
Responsible for Storage + recall of ideas of speech + ideas of complex voluntary motor activity.

Area 17

Function Centre for vision

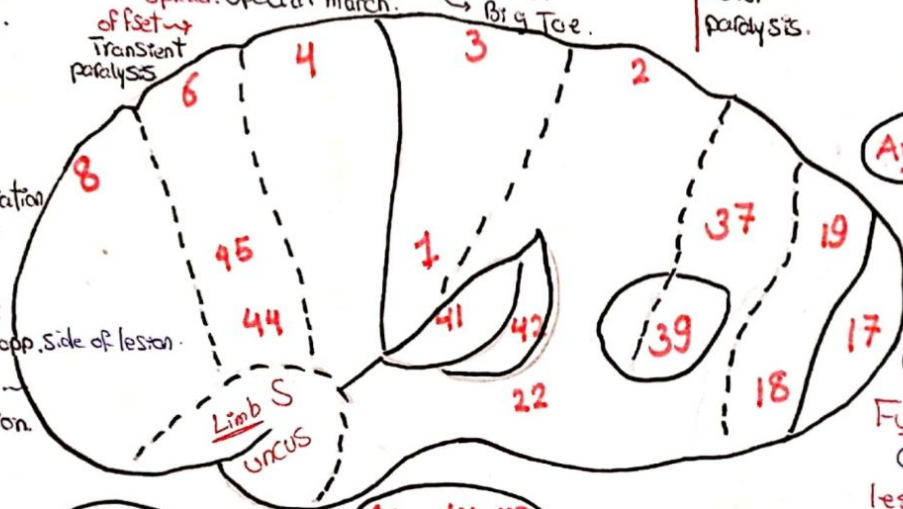
Lesion

Irritative:
visual hallucinations.
Destructive
• contralateral homonymous Hemianopia
• Never complete blindness

Area 18, 19

Function: Recognition and recall of images.

Lesion Visual agnosia
Can see images, but can't recognize them.



Aphasia

Def:

Difficulty or inability of the Formation of speech.

- In absence of lesions of Sense organs (vision - Hearing)
- In absence of mental defect.

Types

1) Sensory Aphasia

"Defect in Comprehension"

a) Visual:

- Visual agnosia lesion in (18, 19)
Patient Sees object, does not recognize them
- Alexia: (lesion in area 39)
Patient can't read as he doesn't understand letters and number. (word blindness).

b) Auditory:

- auditory Agnosia: (area 22)
Patient hears sound but doesn't understand them.

2) Motor Aphasia:

"defect in expression."

- a) Verbal Aphasia: (Broca's area)
Patient can't express himself in spoken words, although he can understand visual and auditory stimuli.

- b) Agraphia: (Exner's area).
The patient cannot express himself in written words, although he can understand letters and numbers.

3) Jargon's Aphasia

"word Salad" defect in association"

- Due to lesion in area 37.
- Patient can speak, But the words are meaningless and not related
- usually associated to Apraxia.

Dysarthria

Difficulty in articulation of speech (è normal formation of speech).

1) Slurred speech:

Def: Disturbance in the production of Consonant especially Labials (B, M) Dentals (D, T).

Causes:

- a) Bilateral Δ lesion "cortico-bulbar lesion" as in pseudobulbar palsy.
- b) LMNL of cranial nerves concerned è speech
 - Nuclear $\rightarrow$ True bulbar palsy
 - Nerve $\rightarrow$ Facial nerve palsy.
 - NMJ $\rightarrow$ MG
 - muscle $\rightarrow$ myopathy (facio-scapulo-humeral)

2) Staccato speech:

Def: Speech is explosive è separation of the syllables.

Causes: Cerebellar lesions

- Hereditary ataxias.
- DS.

3) Scanning Speech (slurred-staccato)

occurs in cerebellar lesions.

4) Monotonous Speech:

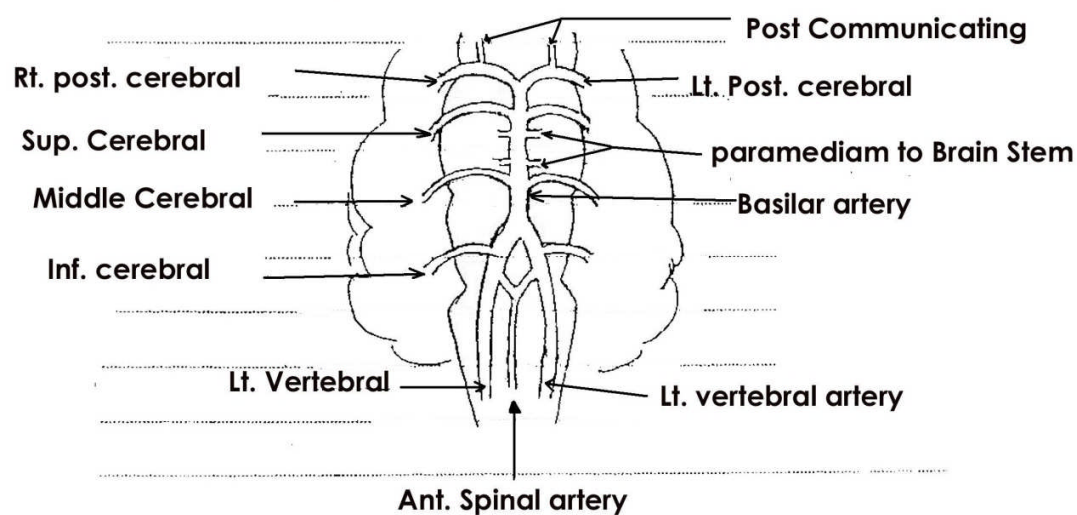
Def: Speech is expressionless and monotonous.

Causes: Extra Δ lesions

- Parkinsonism.

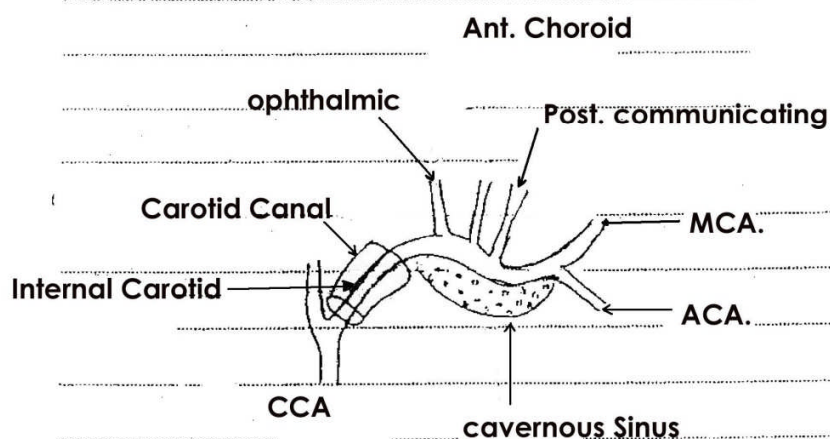
Blood Supply of the Brain

1-Vertebral System



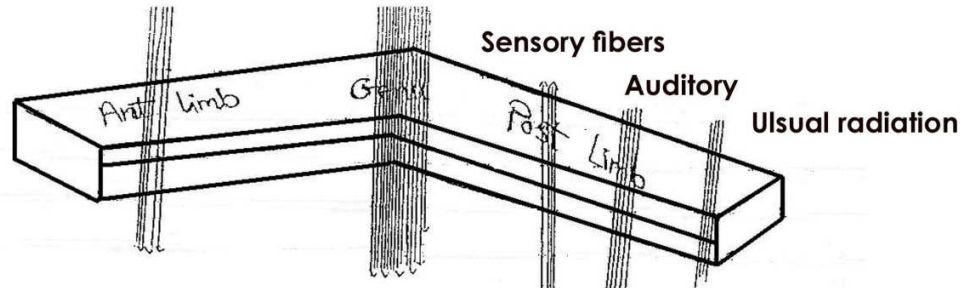
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2- Carotid System

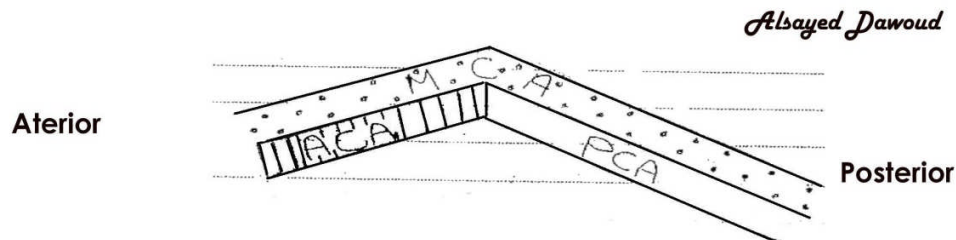


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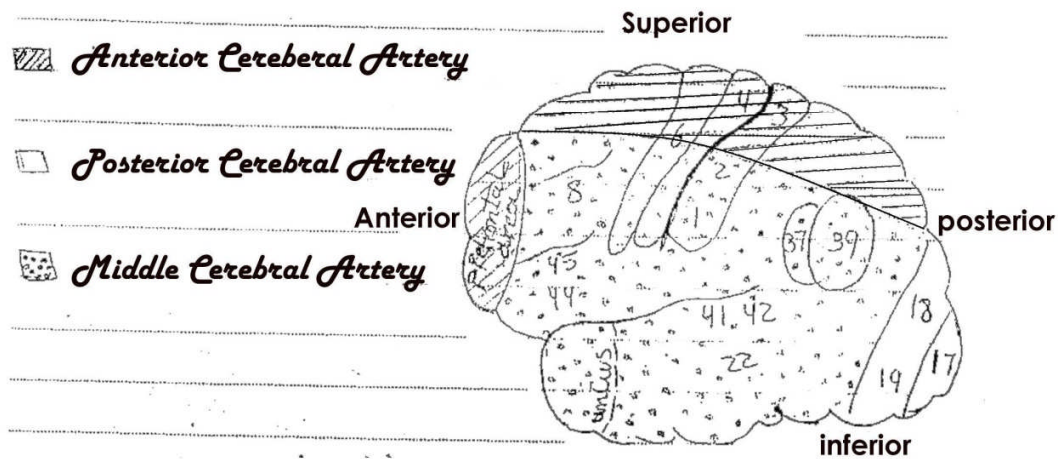
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Internal Capsule



Blood supply of the Internal Capsule



Cerebral Blood Supply

Vertebrobasilar system occlusion

①- anterior spinal artery:

- Quadriplegia if above C5
- Paraplegia if below C5.

②- Brainstem branches :

- Brainstem hemiplegia

③- Posterior inferior cerebellar artery (PICA):

- Ataxia. وتذكر
- Lateral medullary syndrome

- **Sympathetic:** nasal congestion, Horner's syndrome, -ve sternospinal reflex. (No pupil dilatation on pressing sternomastoid)
- **Spinothalamic trunk:** loss of pain & temperature from the opposite side.
- **Cranial nerves:** 10th cranial nerve lesion, palatopharyngeolaryngeal manifestations as (nasal tone, nasal regurge, dysphagia, hoarseness of voice).
- **Vestibular of the 8th cranial nerve :** Vertigo & Vomiting.
- **Spinal nucleus of the trigeminal nerve:** loss of pain & temperature from the same side of the face (crossed hemianaesthesia).

4- Anterior inferior cerebellar artery (AICA):

- Ataxia. وتذكر
- Pass in pons

- **Sympathetic:** as PICA .
- **Spinothalamic trunk:** as PICA .
- **Cranial nerves:** 5th, 6th, 7th >> see cranial nerves.
- **Coclear of the 8th cranial nerve :** Deafness.

5- Superior cerebellar artery: (AICA - 5th, 6th, 7th cranial nerves)

- Ataxia. وتذكر
- sympathetic, spinothalamic, cocclear of 8th cranial nerve:

6- Posterior cerebellar artery:

a- Cortical branch occlusion:

- visual hallucination (area 17) shoulder,
- H. hemianopia
- Visual agnosia (area 18)

b- Deep branch occlusion:

- Thalamus (Thalamic pain, Frozen loss of all sensations)
- Basal ganglia . (extrapyramidal manifestations)

c- Main system occlusion: a+b

Carotid system occlusion**1- Anterior cerebral artery:**a- cortical branch occlusion:

- **Area 1,2,3**(upper part): monohypoesthesia of the paralyzed limb.
- **Area 1,2,3**(lower part): monohypoesthesia of the paralyzed limb.
- **Area 4**(upper part): contralateral monoplegia.
- **Area 4**(upper part): contralateral monoplegia.
- **Area 6**: extrapyramidal manifestations.
- **Area 8**: disturbed conjugated movement of the eye.

prefrontal Area: change personality and mentality.

- **Area 44**: aphasia.
- **Area 45**: agraphia.
- **Area 37**: apraxia.
- **Area 39**: alexia.
- **Area 41,42**: auditory hallucination.
- **Area 22**: auditory agnosia.

b- Deep branch occlusion:

Fascioscapular monoplegia

- Proximal more than distal.
- No sensory loss.

N.B: The only case with proximal muscles affected more than distal muscles as the pyramidal tract lesion cause the opposite.

2- Middle cerebral artery:a- cortical branch occlusion:

- Contralateral upper limb monoplegia.
- Contralateral upper limb monohypoesthesia.

b- capsular branch occlusion: Capsular hemiplegiac- The main system occlusion:

How to answer

Vaso-Occlusive Syndrome

Middle Cerebral is the commonest

1) **Anatomy:**

- ➔ *Origin*
- ➔ *Branches and what the supply*

2) **Causes of occlusion:**

- ➔ *Thrombosis and Embolism* → see Hemiplegia

3) **Clinical picture:**

- ➔ *Cortical*
- ➔ *Capsular*
- ➔ *Main System*

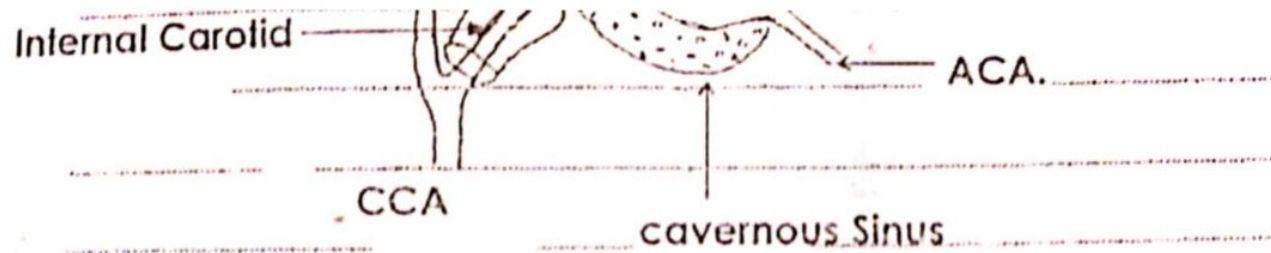
4) **Investigations:** → see Hemiplegia

5) **Complications:** → see Hemiplegia

6) **Treatment:** → see Hemiplegia

Causes of Transient Hemiplegia

- ☑ **T.I.A** (Transient Ischemic Attacks)
- ☑ **Epilepsy**
- ☑ **Aneurysm**
- ☑ **Hysterical**
- ☑ **Hypertensive Encephalopathy**
- ☑ **Migraine**
- ☑ **Multiple Sclerosis**



Circle of willis:

1. Formation:

- 1- Anterior: by union of ant. cerebral Arteries through ant. communicating artery.
- 2- posterior: by union of each post. cerebral artery & the ICA of the same side through the post. communicating artery.

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So, two carotid arteries communicate & each other, and & the VB. system.

2. Site: present in SA. space at the base of brain.

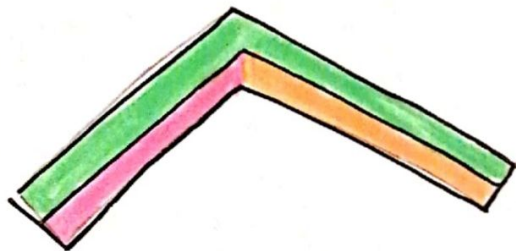
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3. Relation:

- Anteriorly: to the olfactory tract and optic chiasma
- In the middle: To the Cavernous sinus and optic chiasma.
- posteriorly: to the midbrain and oculomotor nerve.

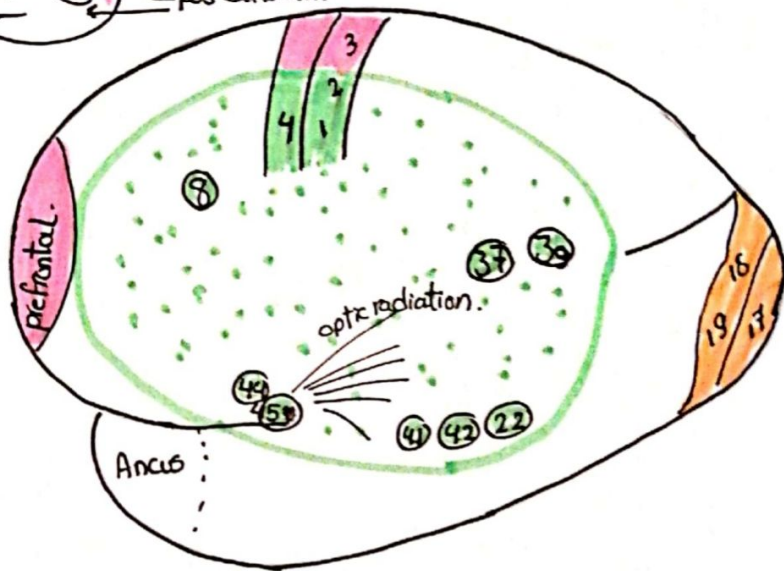
- MCA (Lateral)
- ACA (Medial).
- PCA.

Capsular branches.



corpus callosum.

Cortical branches.



Carotid System

1. MCA occlusion :

a) Capsular : (dorsal 1/2 of IC) (Capsular hemiplegia.)

- 1- Contralateral complete hemiplegia UL/LL.
- 2- " LMNL of facial and hypoglossal nerves
- 3- " hemihyposthesia.
- 4- " Hemianopia may occur.
- 5- No Coma, No convulsion, No aphasia.

b) Cortical

Frontal

- Motor area of face/UL → Contralateral faciobrachial monoplegia
- motor speech area (44) → Motor aphasia
- motor writing area. (45) → Agraphia

Lesion in Dominant Hemisphere.

Parietal :

- Sensory area of UL → Cortical sensory loss of UL (contralateral)
- Angular gyrus (39) → Alexia
- Supramarginal gyrus (37) → Apraxia
- Upper fib. of optic. R. → contralateral lower quadrant hom. hemian.

Lesion in Dominant hemisphere.

Temporal :

- Auditory areas. → Auditory agnosia
- Lower fibers of optic R → cont. upper quadrant hom. hemianopia.

2. ACA :

1- Capsular : (Heubner's A)

Supplies ventral 1/2 of ant. L. of IC.

- Contralateral facio-brachial Monoplegia. (prox. > distal).

2. Cortical



VB. System.

post. cerebral A occlusion

a) Capsular (Thalamo-geniculate A).

Supply → ventral 1/2 of post. L of IC. Thalamus and geniculate bodies

① Thalamia

- 1- Thalamia pain → contralateral.
- 2- Contralateral Hemianesthesia
- 3- Extra Δ manifestation due to ischemia of basal Ganglia.

② LL. monoplegia

b) Cortical. → post 2/5s of Cerebral Hemis

- 1- Contralateral Homonymous Hemianopia + preserved light Reflex + preserved macula
- 2- visual Agnosia (in Dominant Hemis.)

• Frontal branch

- prefrontal Area
- Mentality → mentality changes
- Personality → Personality change
- X of primitive reflex → +ve grasp reflex.

paracentral branch:

- Motor area of LL → cont. Monoplegia in LL
- Sensory area of LL → cont. cortical sensory loss in LL
- paracentral lobule → incontinence of urine (Bilateral Lesion)

Callosal branch

Corpus callosum Apraxia of the same side of domin. hem.

• Frontal branch

- prefrontal Area
- Mentality → mentality changes
- Personality → Personality change
- X of primitive reflex → +ve grasp reflex.

paracentral branch:

- Motor area of LL → cont. Monoplegia in LL
- Sensory area of LL → cont. cortical sensory loss in LL
- paracentral lobule → incontinence of urine (Bilateral Lesion)

Callosal branch

Apraxia of the same side of domin. hem.

Main Arteries occlusion

ICA

etiology: most commonly atherosclerosis.

Age: 4th - 6th decades

Sex: ↑ males

c/p:

Recurrent carotid TIAs:

- 1- Transient Sudden headache
- 2- " Convulsions
- 3- " Ipsilateral Blindness
- 4- " Contralateral Hemiparesis
- 5- " Aphasia (lesion in Domi. hemi)

Stroke (Complete carotid occlusion)

- 1- Ipsilateral Blindness.
- 2- Contralateral Hemiplegia/hemihypothesia
- 3- Aphasia
- 4- cont. homonymous hemianopia.

MCA

- 1- Coma - may be convulsions
- 2- Cont. hemiplegia UL > LL
- 3- Cont. hemihypothesia + more Sensory loss in UL.
- 4- Cont. homonymous hemianopia
- 5- Aphasia + Apraxia

ACA

- 1- Cont. hemiplegia affecting LL > UL
- 2- cont. cortical Sensory loss in LL.
- 3- Mentality and personality changes and +ve grasp reflex
- 4- Incontinence of urine (Bilateral lesion)
- 5- Apraxia of the same side of domi. hemisphere.

VB. Artery.

a) Recurrent VB "TIAs":

- 1- Syncope
- 2- Diplopia
- 3- ophthalmoplegia
- 4- vertigo or tinnitus
- 5- Bulbar Symptoms.
- 6- Hemiparesis, Hemianesthesia
- 7- Ataxia.

b) Stroke (complete occlusion)

- 1- Deep coma
- 2- Complete quadriplegia and decerebrate rigidity
- 3- Bulbar paralysis
- 4- Hypoventilation → Acute RF.

PICA

- 1- acute onset associated è Syncope, hiccup, vomiting, vertigo and pain over the face
- 2- Ipsilateral cerebellar ataxia (Nystagmus, dysarthria, incoordination)
- 3- Ipsilateral Horner's Syndrome.
- 4- " palato-pharyngo-laryngeal paralysis and weakness of Sternomastoid and Trapezius.
- 5- Ipsilateral loss of pain and Temperature Sensations over the face
- 6- Contralateral loss of pain and temperature Sensations over the body.

PCA

- 1- Thalamic Syndrome
- 2- Cont. homonymous hemianopia + preserved Light reflex + preserved macula
- 3- visual Agnosia
- 4- LL Monoplegia.

Def is a broad band of white fibers laying in the depth of the cerebral Hemisphere.

Formed of : 1- Ant. Limb → btn caudate nucleus and lentiform nucleus.

2. Genu

3- post. Limb → btn Thalamus and lentiform Nucleus.

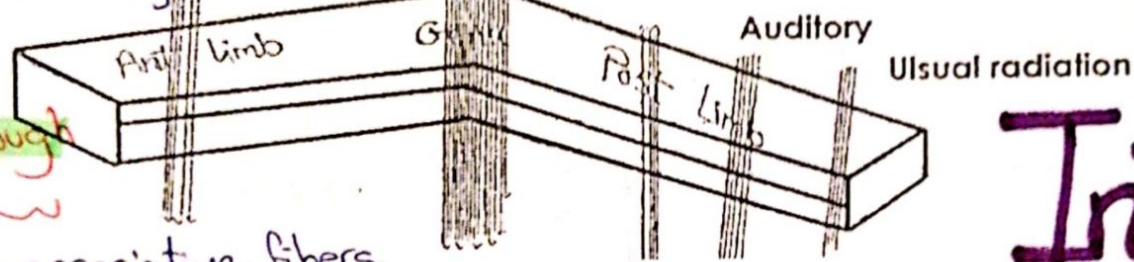
In Capsule Series

Blood Supply : Dorsal 1/2
 ant limb
 knee
 post limb
 } By capsular → lenticulo striate A. branch of MCA. In Capsule series

Neurology

2- ventral 1/2 of ant Limb → By Capsular branches of ACA (Heubner's artery).

3- ventral 1/2 of
 knee
 post limb
 } By capsular branches of PCA (Thalamo-geniculate A). Sensory fibers



Internal Capsule.

AL → ant. part → associative fibers.
 → adjacent part to genu → pyramidal fibers.

Internal Capsule

PL → itself → Sensory fibers from thalamus. (ascend)
 → ant 1/2 → pyramidal fibers (descend)
 → post. part → auditory + optic radiation

Alsayed Dawoud



Genu → pyramidal fibers

Blood supply of the Internal Capsule

→ Supply arm are followed by those supplying head, trunk and lastly LL.

Superior

Stroke

Definition :

- A sudden neurological deficit of vascular origin that persists for longer than 24 hours or leading to death.
- Stroke is the third most common cause of death, after coronary heart disease & cancer.

Types & causes :

I. **Ischemic** : (Cerebral infarction) *85% of all strokes.*

a) Thrombosis :

- ➞ Atherosclerosis : The most common cause
 - ✓ Extracranial : aorta , carotid or vertebral
 - ✓ Intracranial : ACA , MCA , PICA
- ➞ Arteritis , Dissection.
- ➞ Hematological disorders : Polycythemia, leukemia, sickle cell anemia & hypercoagulability state.

b) Embolism :

- ➞ From the heart :
 - ✓ Atrial fibrillation (AF)
 - ✓ Infective endocarditis.
 - ✓ Mitral Stenosis
 - ✓ Myocardial infarction.
 - ✓ Atrial myxoma
 - ✓ Dilated cardiomyopathy
 - ✓ Congenital heart disease.
- ➞ From carotid vessels

Risk factors for ischemic stroke

- Risk factors of atherosclerosis : modifiable & non modifiable see cardiology
- Transient ischemic attacks.

II. Hemorrhagic : *accounts for 15% of all strokes*

- a. **Intracerebral** : 2 HAT *see hemiplegia*
- b. **Subarachnoid** hemorrhage. 2A , 2H , 2T

Clinical feature of ischemic stroke :

1- Focal neurological manifestations : according the occluded artery

*Clinical features of occlusion of the **MCA** or its branches:*

Cortical branch occlusion : Supplies the lower 3/4 of the anterior 3/5 of CC

- Contralateral UL monoplegia with cortical sensory loss.
- Other cortical manifestations : aphasia , agraphia , apraxia (If the left hemisphere is involved)

Capsular branch occlusion : capsular hemiplegia 6 criteria P4

*Clinical features of occlusion of the **ACA** or its branches:*

Cortical branch : supplies upper 1/4 of the anterior 3/5 of the cerebrum

- Contralateral monoplegia in LL with cortical sensory loss
- Mentality & personality changes.

Capsular branch : supplies ventral surface of the anterior limb of the internal capsule.

- Contralateral facio-brachial monoplegia.

Vertebro-basilar system : supplies the posterior 2/5 of the cerebrum , the cerebellum , the brain stem & the spinal cord.

1- anterior spinal artery occlusion:

- ☠ Quadriplegia if above C5
- ☠ Paraplegia if below C5. (complete flaccid paraplegia)

2- Brainstem branches occlusion : Brainstem hemiplegia

3- Posterior inferior cerebellar artery (PICA):

- ☠ Ataxia. (.....)
- ☠ It is NOT associated with hemiplegia.
- ☠ Lateral medullary syndrome (*Wallenberg syndrome*) : 🖐



1- Sympathetic: Horner's syndrome :

- Ptosis - Myosis - Anhidrosis - Enophthalmos - Nasal congestion

2- Spinothalamic tract : loss of pain & temperature from the opposite side.

3- Cranial nerves: 10th cranial nerve lesion → palatopharyngeolaryngeal manifestations as (nasal tone, nasal regurge, dysphagia, hoarseness of voice).

4- Vestibular of the 8th cranial nerve : Vertigo & Vomiting.

5- Spinal nucleus of the trigeminal nerve: loss of pain & temperature from the same side of the lesion (*crossed hemianaesthesia*).

Stages : Flaccid then spastic stage P3

2- Negative in quality :

Loss of function rather than positive (e.g. *Paralysis rather than jerking , blindness rather than visual hallucination*)

3- Rapidly developing : The onset is sudden and acute.

4- Maximum at onset : Over minutes to hours.

5- 80% of stroke patients have at least one vascular risk factor and most are elderly. Stroke is uncommon (but not rare) in young people.

Investigations of ischemic stroke

1- First line investigations :

- CBC & ESR.
- INR , PTT
- Plasma glucose , cholesterol, urea , electrolytes.
- Urinalysis.
- ECG.

2- Cerebral imaging :

i. CT brain :

- To exclude non vascular causes e.g. tumor.
- To differentiate an ischemic stroke (hypodense blackish lesion) from hemorrhagic stroke (hyperdense whitish lesion)

ii. MRI : Usually detects area of evolving infarction within hours, CT is sometimes negative for up to several days after acute infarction.

3- Echocardiography : if cardiogenic embolism is suspected.

4- Duplex carotid ultrasound : When carotid artery occlusion is suspected.

5- Angiography : it plays an important role in the preoperative evaluation of carotid artery disease.

Treatment of ischemic stroke

~~✍~~ **As in hemiplegia : 6 items P7 plus**

~~✍~~ **Carotid endarterectomy for patient with severe carotid stenosis.**

1- General care of the comatosed patient : 🖐 b

2- Symptomatic ttt : 🖐

3- Rehabilitation :

- Physiotherapy
- Speech therapy.

4- Control of blood pressure : Antihypertensive therapy is indicated in severe hypertension ($\geq 220/120$) or in those with hypertensive encephalopathy, aortic dissection, acute pulmonary edema or myocardial infarction.

5- Specific treatment :

- ~~✍~~ Thrombolytic therapy
- ~~✍~~ Anticoagulants
- ~~✍~~ Anti-platelets

6- Neuroprotective.

Indications of thrombolytic therapy in stroke :

- Acute ischemic stroke with clearly definable time of onset.
- 3 hours time window. (time is brain)
- A baseline CT excluding ICH & SAH.
- Age is 18 years or older.
- Dose of i.v. alteplase (tissue plasminogen activator) : 0.9 mg/kg (max. 90 mg) infused over 1 hour.

Absolute contraindications : 11

- CT scan today shows intracranial bleeding.
- Symptoms suggestive of subarachnoid hge, even if the CT is normal.
- CT scan today shows multilobar infarction (hypodense area > $\frac{1}{3}$ of cerebral hemisphere.
- Prior history of intracranial bleeding.
- Any of the following within the past 3 months : intracranial or intraspinal surgery, serious head trauma.
- Blood pressure > 185 mmHg (systolic) or > 110 mmHg (diastolic)
- Evidence of active internal bleeding.
- Patient has an arteriovenous malformation or aneurysm.
- Lab evidence of coagulopathy : e.g. platelets count < 100000
- Patient received heparin in the past 48 h and PTT above normal range
- Patient received warfarin & INR > 1.7

Relative contraindications : 4

- Major surgery or serious trauma in past 2 weeks.
- GIT or urinary tract bleeding within past 3 weeks.
- Acute MI in past 3 months or post MI pericarditis.
- Blood glucose < 50 or > 400 mg/dl

Prognosis

Complete recovery is uncommon, but the sooner improvement begins, the better the prognosis.

Depends on :

- Site & size of infarction.
- Patient age
- Other comorbidities.
- Impaired consciousness , aphasia , or severe brain stem signs suggest poor prognosis.

Hemorrhagic stroke

Types & Etiology :

- 1) **Intracerebral hemorrhage** : See above 2H, 2A, 2T
 - Primary intra-cerebral hemorrhage : intracerebral hemorrhage due to chronic hypertension. "hypertensive ICH"
 - Chronic hypertension produces fibrinoid necrosis, lipohyalinosis, and medial degeneration, which make the vessels susceptible to rupture.
- 2) **Subarachnoid hemorrhage** : 2A , 2H , 2T

Clinical picture of intracerebral hemorrhage :

- 1) ICH occurs characteristically during activity (rarely during sleep).
- 2) Symptoms typically begin abruptly.
- 3) The clinical presentation has 2 elements :
 - a) Increased Intracranial pressure : headache , vomiting , loss of conscious in some cases. Seizures are uncommon at the onset in 7% of cases but are found in 32% of lobar hemorrhages.
 - b) Specific clinical manifestations : which are related to the site of hematoma.

4) In order of frequency, the most common site of primary cerebral hemorrhage are :

- 1- Putamen. (*a basal ganglia nucleus*)
- 2- Lobar hemorrhage : central white matter of frontal, parietal, temporal & occipital.
- 3- Thalamus.
- 4- Cerebellum.
- 5- Pontine hemorrhage.

1- Putamen :

- This is the commonest type.
- Clinical manifestations vary with the size of hemorrhage, but include hemiparesis, headache, and alterations of consciousness.

2- Lobar hemorrhage : Headache is a first and most prominent symptom.

- Frontal hematoma : hemiparesis more in upper limb
- Parietal hematoma : sensorimotor deficit
- Dominant temporal hematoma : fluent aphasia
- Occipital hematoma : homonymous hemianopia.

3- Thalamic hemorrhage : 15%

- hemiparesis or hemiplegia in nearly all cases with severe sensory deficit
- Aphasia may be present with lesion of the dominant side.
- Extension into the subthalamus & high midbrain may cause ocular disturbances (downward deviation of the eyes & irreactive pupil)
- Rupture into the 3rd ventricle may lead to early hydrocephalus.

4- Cerebellar hemorrhage : 10%

- Acute onset of headache , vertigo , vomiting & inability to stand and walk.
- characteristic syndrome of ipsilateral ataxia, LMN facial palsy, and ipsilateral gaze paresis
- Coma & death may occur due to brainstem compression.

5- Pontine hemorrhage : 6%

- Characterized by coma, quadriplegia, absence of light reaction, pinpoint pupil , decerebrate rigidity, respiratory disturbance, tachycardia, hyperthermia. Death usually occurs within few hours.
- Less severe cases arising from hemorrhage confined to one side of the pons are also recognized.

Diagnosis of intracerebral hemorrhage

1) Clinical feature : suggested by :

- Acute hypertension exceeding the patient's chronic hypertensive level.
- Vomiting at onset.
- Severe headache.

NB : Although the absence of headache or vomiting does not rule out the diagnosis of ICH, presence of these symptoms is in favor of ICH or SAH as it is reported in less than 10% of occlusive strokes.

- Focal seizure.
- Hypertensive changes of the retina.

2) Brain CT : is the procedure of choice to detect the intracerebral hemorrhage (white mass) with surrounding brain edema.

3) MRI.

4) Lumbar puncture : must performed cautiously as it may precipitate or aggravate an impending cerebellar herniation.

5) Others : PT , PTT , Platelets count , Liver function tests.

Prognosis

- 1/3 of the patient die in the first 1 to 30 days
- Death occurs due to either :
 - 1) Marked rise of intracranial pressure $\Rightarrow$ $\downarrow$ brain perfusion.
 - 2) Compression of vital centre such as hypothalamus , brainstem.

NB : Patients who survive usually show a surprising degree of restoration of function , since in contrast to infarction the hemorrhage pushes the brain tissue rather than destroys it.

Treatment : see hemiplegia

1- Intracerebral hemorrhage :

- As ischemic infarction but **surgical evacuation of blood** instead of thrombolytic & anticoagulant therapy.
- **Antihypertensive therapy** is initiated when the systolic blood pressure is > 180 mmHg or the diastolic blood pressure > 105 mmHg.

2- Subarachnoid hemorrhage : see later p51

Transient ischemic attacks (TIAs)

Definition :

- TIAs are short transient attacks of cerebral ischemia lasting less than 24h
- Most TIAs last only 10 seconds to 15 minutes.
- RIND (Reversible Ischemic Neurological Deficit) : If the attack lasts over 24 h .
- Patients with TIAs have a higher risk of developing a stroke.

Etiology : The same as ischemic infarction

- Cerebral thrombosis.
- Cerebral embolism.

Clinical picture :

- Carotid TIAs : see before
- Vertebro-basilar TIAs : see before

Investigations : The same as ischemic stroke

Treatment :

I. Medical :

- Antiplatelets : Aspirin , plavix.
- Anticoagulants : Heparin , Warfarin.
- Neuroprotective : **Trental** , **Trivastal**.
- Treatment of risk factors : e.g. hypertension , DM, hyperlipidemia, smoking.

NB : LDL goal < 70, TG goal < 150 & glucose concentration of 140-180 mg/dL

II. Surgical :

Endarterectomy : An incision is made to open the carotid artery , the plaque is removed.

III. Carotid angioplasty : Introduction of balloon to dilate the stenotic artery.

Paraplegia

Definition :

Paralysis of both lower limbs due to bilateral pyramidal tract lesion (UMNL).

Etiology :



1. **Cerebral :** (cerebral lesion involving both cortical leg areas), rare
 - Vascular : Occlusion of unpaired anterior cerebral artery, Superior sagittal sinus thrombosis.
 - Trauma : depressed fracture of the vault of the skull.
 - Tumor : Parasagittal meningioma.
 - Cerebral palsy.
2. **Brainstem :** midline lesions
 - Syringobulbia (fluid-filled cavity within the brainstem).
 - Midline tumor e.g. glioma.
3. **Spinal paraplegia :** The most common
 - I. **Focal :**
 - a) **Spinal cord compression :**
 - Vertebral : “**Extramedullary**” :
 - Trauma : accidents, gunshot, pathologic fractures due to rheumatic diseases.
 - Tumor : primary or metastatic spinal tumors.
 - Infection : Spinal tuberculosis (Pott’s disease) is the most common chronic infection causing paraplegia.
 - Meningeal : “**Extramedullary**” : Meningitis, meningioma, leukemia & lymphoma
 - Cord : “**Intramedullary**” : Syringomyelia (cavitation of the cord), glioma.
 - b) **Inflammatory :** Transverse myelitis.
 - c) **Vascular :** Anterior spinal artery occlusion.

- II. **Multifocal (Disseminated) :** Disseminated sclerosis (DS)
- III. **Systemic :**
 - Idiopathic : Motor neuron disease (MND)
 - Hereditary :
 - Hereditary spastic paraplegia (HSP)
 - Friedreich's ataxia, Marie's ataxia.
 - Subacute combined degeneration of spinal cord : due to vitamin B₁₂ deficiency.
 - **Tropical spastic paraparesis** : caused by viral infection (Human T lymphotropic virus).

Clinical picture :

1) At the level of lesion : In a case of focal paraplegia

- A. **Vertebral manifestations :** Localized pain, tenderness, swelling, deformity.
- B. **Radicular manifestations :** Only in extramedullary paraplegia.
 - Posterior root : girdle pain & later hypoesthesia in the dermatome supplied by the affected root.
 - Anterior root : LMNL in the muscles supplied by the affected root (usually trunk & abdominal muscles).

2) Below the level of lesion (cord manifestations) :

- Clinical manifestations are classified into **3 groups** :

1. Motor manifestations :



a) Flaccid paraplegia (spinal shock stage) : *see hemiplegia ... wait ... wait see here, baby*

- Occur only in cases with **acute onset** (as trauma, Transverse myelitis & anterior spinal artery occlusion).
- Initially, the paralysis may show the criteria of **LMNL** (hypotonia, hyporeflexia) but the planter response is extensor.
- This stage lasts for about 4-6 weeks & may persist longer in some cases. The more the prolongation of this stage, the worse the prognosis.

b) Spastic paraplegia (Paraplegia in extension) :

- The paralyzed muscles show the criteria of **UMNL** (hypertonia, hyperreflexia,...)
- **Extensors** of LL are more spastic than flexors, hence the name Paraplegia in extension.

c) Paraplegia in flexion :

- With further progression, the extrapyramidal fiber in the cord will be affected.
- Increased tone in the **flexors**, *hence the name*.
- There are 2 reflexes in this stage : Mass reflex & Piere Marie Foix reflex.

Mass reflex : Scratching of the medial aspect of the thigh, results in flexor muscle spasms, micturition , erection, hypertension and profuse sweating. Nurses should avoid stimulating this area. 😊

Piere Marie Foix reflex : Passive plantar flexion of the toes causes dorsiflexion of the ankle and flexion of the knee and hip.

2. Sensory manifestations :**a) Extra-medullary (vertebral or meningeal) compression :**

- There is a **sensory level** which approximate the level of injury.
- Below this level, **ALL** sensations are lost.
- Early loss of sensation in the **saddle area**.

b) Intramedullary (cord) compression : e.g. syringomyelia

- Double sensory levels (Jacket sensory loss) with normal sensations above and below it.
- Dissociation of sensation 🎵 : Loss of pain & temperature but preservation of touch & deep sensations.
- There is usually sacral sparing. Why baby? Since the ascending spinothalamic fibers from the sacrum lie peripherally & tend to be the last that are affected by the expanding cavity.

3. Bladder manifestations :

- **Retention of urine** in **acute lesion only** e.g. trauma followed by automatic bladder.

Why ?

- The initial neurologic response is spinal shock stage (flaccid paralysis). So, the bladder wall is paralyzed & the external sphincter is still normal.
- During this time, the urinary bladder must be drained with CIC (Clean Intermittent Catheterization) or indwelling urethral catheter.

- Complete lesion : **Automatic bladder** (Voluntary inhibition of the micturition reflex is lost).
- Incomplete (partial lesion) : disturbance of voluntary micturition
 - Precipitancy : difficulty in inhibiting the act.
 - Hesitancy : difficulty in starting the act.
 - Urgency : sudden and strong desire to urinate.
 - Incontinence.

- The rectum : similar of that of the bladder but less severe.
- Impotence : may be caused by bilateral UMNL or LMNL.

Investigations :

1. Plain X-ray of the spine : may reveal the underlying cause in a case of vertebral compression e.g. trauma, Pott's disease, spondylosis...
2. CT & MRI : **MRI is the preferred method** of investigation of the spinal cord disease.
3. CSF examination : is of limited value

In Extramedullary compression : there is marked increase of proteins leading to yellowish discoloration, spontaneous coagulation & cyto-albuminous dissociation

"Froin's syndrome". 🎵🎵

4. **Myelography** : radiography after injection of a water soluble radio-opaque dye into the subarachnoid space. Normally, the dye drops down to the level of S₂ vertebra.

○ In Extramedullary compression :

arrest of the dye at the level of compression “saddle shape”.

- In intramedullary compression : partial arrest of the dye “fusiform shape” .

5. Others : CBC, ESR, Ca, liver function tests....

Spinal cord levels relative to the vertebral bodies :

- The spinal cord has 31 segments. (8 cervical, 12 thoracic, 5 lumbar, 5 sacral, 1 coccygeal).
- It ends at the first lumbar vertebral body.
- Spinal cord segments are not always situated at the corresponding vertebral levels.

Spinal cord level	Corresponding vertebral body
C1 – C7	Same as cord level
C8	-1 (exits below C7 vertebra)
T1 - T6	-2
T7 - T12	-3 (so, T12 ⇔ situated in T9)
L1, L2	T10
L3, L4, L5	T11
S1, S2	T12
S3, S4, S5	L1

There are 8 cervical segments & only 7 cervical vertebra.

Treatment :

I. General nursing care :

- **Bed**: frequent change in the position to prevent bedsores.
- **Bladder** : urinary catheter.
- **Bowel** : daily enema.
- **Breathing** : (care of respiration)
- **Balanced nutrition**.

II. Symptomatic treatment :

- a) Analgesia for pain.
- b) Sedative : Benzodiazepine (midazolam) may be given.
- c) Muscle relaxants for spasticity.

III. Physiotherapy :

- Regular passive & active exercise of the paralyzed limbs.
- Positioning of the paralyzed limbs in the optimal position.

IV. Specific treatment : Surgical or medical according to the etiology.

- Anti-tuberculous drugs for pott's disease.
- Dexamethasone : 10 mg IV followed by 4 mg IV/ 6h for compressive lesions & transverse myelitis.
- Surgery : for spinal cord compression.
- Radiation combined with high dose steroid : for cord compression due to malignancy.

Special forms of paraplegia

	Transverse Myelitis (TM)	Syringomyelia
Def.	acute infl. affecting gray and white matter in one or more adjacent spinal cord segments → Common in Thoracic Seg.	Chronic disorders characterised by 1-Lesion → Long cavities surrounded by gliosis 2-site: in central part of gray matter of S. cord Common in → Lower cervical, upper thoracic
Etiology	1- Idiopathic 2- infection. Viral: Bacterial: 3- immunologic: Following vaccination • associated w/ vasculitis eg SLE 4- Iatrogenic and toxic • post lumbar • IV. heparin. 5- Demyelinating: DS, DEM.	• Congenital • Idiopathic
C/p.	Shock stage: - Paraplegia & flaccid paraplegia + Sensory level below which all types of Sensations are lost - sphincteric: Retention of urine & overflow Recovery stages paraplegia: Spastic + Sensory level loss below which all types of Sensations are lost sphincteric: Precipitancy of micturation. DD:	Age → b/n 15-35 years onset-course → gradual onset - slowly progressive course ① Cervical Syringomyelia: 1- Motor: early in LL: Localized LMN weakness + fasciculation due to encroachment on AHCs late in LL: Spastic paraplegia (UMN). 2- Sensory: Jacket sensory loss of dissociated nature. + Sacral spare 3- Autonomic: - Horner's \$ - Morvan's \$ 4- Associated skeletal anomalies. Des Cavus / Spina bifida ② Lumbar Syringomyelia: as epiconus lesion p. 21.
Inv.	CSF exam: • ↑ both cells, ptns • In DS → ↑ Igs. MRI: to exclude acute compression on cord.	- MRI * (cavity) - Myelography. (obsolete).
Ttt.	1- Non-specific symptomatic Ttt 2- Corticosteroids. • prednisone in Idiopathic, SLE, DS • ACTH (IN) in <u>DS</u>	1- X-ray irradiation of the affected region of the cord 2- physiotherapy + Symptomatic Ttt.

Cauda Equina

Definition: Collection of lumbosacral roots in lower part of spinal canal.

Conus medullaris : the last 3 sacral segments of spinal cord (S3,4,5)

Epiconus: the above 4 segments of spinal cord (S1,2-L4,5)

Etiology:

1. Congenital → Spina bifida.
2. Trauma → fr. Dislocation of lumbar vertebrae .
3. Tumor → meningioma & neurofibroma.
4. Inflammation → Pott's disease .
5. Degeneration → lumbar spondylosis .

Clinical picture:

1) Motor: 1st convulsions then paralysis or paresis of muscles supplied by the same root;

# L2	- Flexion of hip	- Iliopsoas
# L3	- Extension of Knee	- Quadriceps
# L4	- Dorsiflexion of ankle	- Ant. Tibial group
# L5	- Dorsiflexion of toes	- Ant. Tibial group & Glutei
# S1	- Planter flexion ankle & toes	- Calf & Glutei
# S2	- Flexion of knee & extension of hip	- Hamstrings
# S3,4,5	- Anal contraction	- Anal & Perianal muscles

2) Sensory:

Pain and **parasthesia** in the area supplied by the affected root → then loss of the superficial and deep sensations in the area supplied by the affected root.

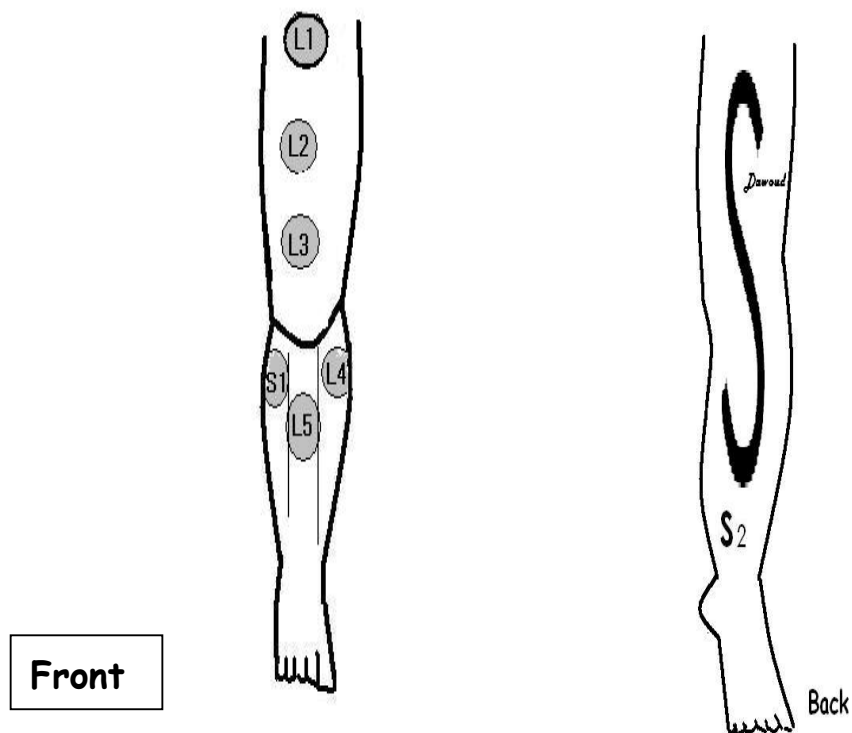
- ☒ **L1** → Upper 1/3 of front of thigh.
- ☒ **L2** → Middle 1/3
- ☒ **L3** → Lower 1/3
- ☒ **L4** → Anterolat. Aspect of the thigh , front of knee , Anteromedial aspect of the leg & medial aspect of dorsum of foot and big toe
- ☒ **L5** → Middle 1/3 of front of leg , dorsum of foot & middle 3 toes
- ☒ **S1** → Posterolat. Aspect of thigh and leg , lat. 1/3 of dorsum & little toe
- ☒ **S2** → Back of thigh and leg & sole of foot
- ☒ **S3,4,5** → Anal and perianal & gluteal region (Saddle shaped area)

Conus:

- ☑ Early autonomic bladder and fecal incontinence
- ☑ Impotence
- ☑ Impairment of sensations in saddle shaped area

Epiconus:

- ☑ Weakness or paralysis in muscles supplied by L 4,5 & S1,2
- ☑ Ankle reflexes absent BUT Knee reflexes are intact
- ☑ Sensory loss from L4 to S2
- ☑ Bladder disturbance (*may occur in the form of pericetancy*)



3) Bladder manifestations:

See neurogenic bladder

Spondylosis:

Def: Gradual, progressive degeneration of intervertebral discs, specially those which are freely mobile $\xrightarrow{\text{wear Tear}}$ Cervical, Lumbar.

PF:

1. old ages
2. occupational $\rightarrow$ Labourers.
3. DM: Degeneration of Annulus fibrosis.

pathology: intervertebral disc is formed of $\rightarrow$ Central gelatinous (Nucleus pulposus) $\rightarrow$ Fibrous tissue ring (annulus fibrosus)

Disc Degeneration:
Degeneration of annulus fibrosus $\rightarrow$ Herniation of nucleus pulposus $\rightarrow$ Compression of adj. structure
weakest parts of annulus fibrosus $\rightarrow$ Lat, post.
Herniation: Lateral, posterior or posterolat.

Associated Degeneration:
• Sclerosis of adjacent surfaces of vertebrae
• Lipping or osteophyte formation due to calcification of prolapsed parts and Ligament

C/P

1- C/P of cervical Spondylosis

Important Signs.

- Hoffman Sign: reflex contraction of thumb and index after nipping of middle finger
- Lhermite Sign: Electric-like sensation felt in the back and limbs on bending the neck is due to PC affection in the cervical region.

presentation

1. Features of root compression.

- a) anterior root compression. (Lateral prolapse) LMN paralysis
- b) post root compression. (brachial Neuralgia)
Early $\rightarrow$ pain-parathesia
late $\rightarrow$ Hypothesia.

Root	Action	Muscle	Root	Sensory disturbance:
C _{1,2}	Lateral movement of neck	Sternomast, Trapez.	C ₂	Lat. Aspect of neck
C _{3,4}	Elevation of shoulder.	Supra, infra spinatus	C _{3,4}	Shoulder down to manubrium st. anteriorly.
C ₅	Abduction of shoulder.	Deltoid	C ₅	Lat. aspect of arm.
C _{5,6}	Flexion of elbow	Biceps, brachioradi.	C ₆	lat. aspect of forearm, thenar eminence, thumb.
C _{6,7}	Extension of elbow	Triceps.	C ₇	middle aspect of forearm, middle of palm, middle 3 fingers.
C _{7,8}	Extension of wrist.	Extensors of wrist	C ₈	medial aspect of forearm, hypothenar eminence and little finger.
C _{8,T₁}	Flexion of wrist and movement of small muscles of hands. (post. prolapse)	Flexors of wrist	T ₁	Medial aspect of arm.

2. Features of Cord Compression:

- a) of ΔT
- weakness or paralysis
 - Signs of UMN below level of compression
 - Bladder disturbance: Rare and late
- b) of spino-thalamic
- Superficial Sensory loss below level of compression (pain - Temp)
- c) post. column
- Deep Sensory loss ie. Sensory ataxia below level of compression

3. Features of root and cord: (postero-lateral prolapse).

In ULs: Combined Signs of LMNL in the form of wasting of muscles + Signs of UMN in the form of Hypertonia and hyperreflexia (Tonic atrophy).

In LLs weakness $\hat{=}$ Signs of UMN.

II c/p of Lumbar Spond:

As Cauda Equina lesion.

Inv:

I - MRI, CT scan.

- MRI is the imaging of choice
- will detect small disc prolapse.

2 - plain Xray.

- Narrowing of Intervertebral disc spaces.
- Sclerosis of adjacent surfaces of the vertebrae.
- Lipping or osteophyte formation.
- Straightening of The spine (loss of lordosis).

Treatment:

I - Medical:

- Analgesics : NSAIDs, vit B complex
- Ms relaxants.

II - physiotherapy:

- Short wave Therapy
- plastic neck collar. (max 3 months)
To avoid Ms Fibrosis.
- Traction of vertebrae

III Surgical

Decompression by laminectomy.

Indications:

- Failure of medical treatment.
- Severe intolerable pain.
- Sensory or motor affection.
- Bladder disturbances

Neurogenic Bladder

Definition:

Sensory, Motor and Autonomic manifestations due to impaired nerve supply of urinary bladder.

Control of act of micturition:

- ✎ The wall of urinary bladder contain stretch and chemical receptors which are stimulated by fullness of bladder and acidity of urine .
- ✎ Efferent impulses are carried by S2,3,4 sensory parasympathetic fibers to 2nd, 3rd and 4th sacral segments of spinal cord .
- ✎ Efferent impulses carried by S2,3,4 motor parasympathetic fibers lead to contraction of the wall and relaxation of the sphincter → Evacuation (in infant)
- ✎ The Afferent impulses reaching the sacral part of the cord ascend in the post. Column → Brain where sensation of full bladder is perceived.
- ✎ Efferent impulses descend from a special bladder cortical center for the conscious control of micturition.

*This center → on the medial surface of cerebral hemisphere in front of foot area.
→ is developed with myelination of the pyramidal tract after the age one year.*

- ✎ Efferent impulses (inhibitory impulses) descend from this center to 2nd, 3rd and 4th sacral segment through pyramidal tract.
- ✎ Micturition takes place normally when this inhibitory impulses are released

Supraspinal inhibitory impulses are absent in infants below one year due to Absence of myelination of pyramidal tract and immaturity of the cortical bladder center.

Lesions:

1) Afferent lesion :

Over urinary bladder filling with urine → gradual enlargement
(pr. of urine > pr. of sphincter)→ Dripping ## Sensory atonic bladder ##

2) Efferent lesion:

Mild urinary bladder filling with urine → Inability to evacuate voluntarily →
Cauterization. ## Motor atonic bladder ##

3) Center lesion:

NO nerve supply to urinary bladder → evacuation by myogenic contraction
Autonomic bladder ## (Involuntary & Irregular & Incomplete.)

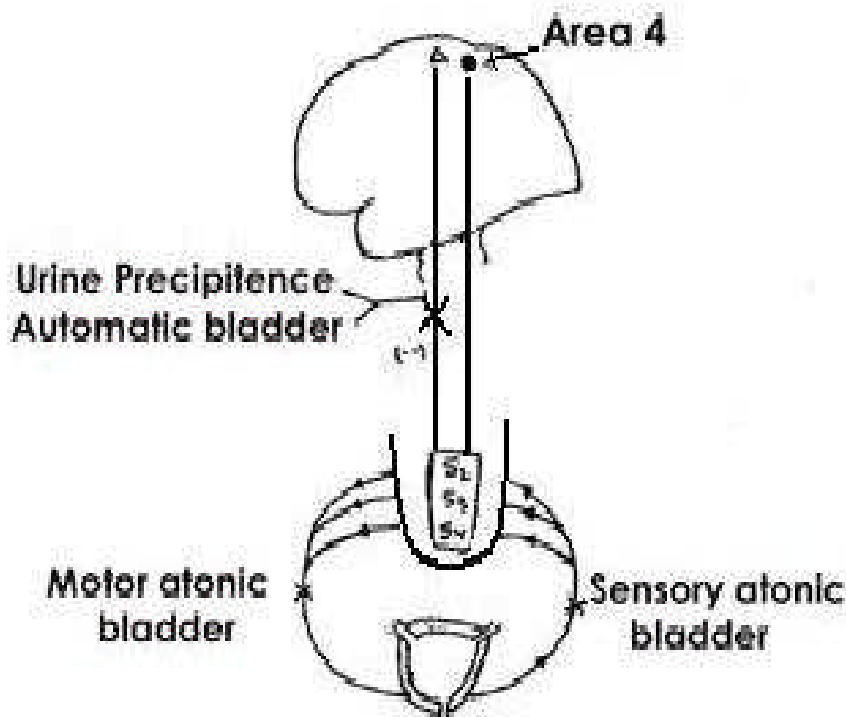
4) Pyramidal lesion:

☑ **Acute lesion:** Retention of urine

☑ **Gradual lesion:**

a) *Complete*: ## Automatic bladder ## (involuntary & regular & complete)

b) *Partial*: Precipitancy of urine.



Peripheral neuropathy

Definition: Degeneration of peripheral nerves.

Classification:

1) Anatomical

- A. **Mononeuropathy**, affecting ONE nerve in ONE limb.
- B. **Mononeuropathy Multiplex**, affecting > one nerve in ONE limb
- C. **Polyneuropathy**, affecting pr. Nerves of ALL limb

2) Aetiological

- A. **Mononeuropathy**, (DM.&Leprosy &Polyarthritis Nodosa&Sialica)
- B. **Mononeuropathy Multiplex**, (DM.&Leprosy &Polyarthritis Nodosa&Sarcoidosis&Amyloidosis)
- C. **Polyneuropathy**, (Congenital& Acquired)

Manifestations:

➤ MOTOR:

1. Paralysis of muscles with LMNL (and mention its 5 criteria)
2. Bilateral and Symmetrical *as it is systemic*
3. Affect distal muscles > Proximal
4. Affect extensors > flexors
5. Affect LL > UL
6. Affect ankle reflex > knee reflex
7. Affect **1673** nerves

N.B: the more the length of the nerve, the more ↓ its nutritional supply from the cell,

The more weakness and motor manifestations.

➤ SENSORY:

1. Glove and stock hyposthesia.
- 2.

	<i>Superficial Sensations</i>	<i>Deep Sensations</i>
<i>components</i>	<i>-Pain - Touch -Temp.</i>	<i>-Muscles</i>
<i>Irritation</i>	<i>-Pain - Parasthesia</i>	<i>-Tender muscle</i>
<i>Destruction</i>	<i>-Lost superficial sensations</i>	<i>-Lost deep reflex - Sensory ataxia</i>

Autonomic: See Diabetic neuropathy. (*Endocrine book*)

CONGENITEAL

☞ Proneal muscle atrophy

- # Inverted champagne bottle appearance.
- # Sever muscle wasting with mild weakness.

☞ Refsum's disease

* decrease in hydroxylase enz. → increase in phytanic acid → 3S

1. Cerebellum: ATAXIA
2. Skletal deformity
3. Special senses: BLINDNESS&DEAFNESS

*ttt. Plasma Phoresis

Classification or Etiology of

Polyneuropathy

AQUIRED

3 I & 2 T & MEN

1. **Metabolic:** DM. & Any failure { DM. affect 3rd & 4th & 6th cranial Nerves} MCQ
2. **Endocrine:** all
3. **Nutrition:** vit.B complex deficiency
4. **Toxins:** ⇒ Endo.(LCf.& RF.)
⇒ Exo. (Heavy metals)
5. **Tumor:** Paramalignant \$.
6. **Iatrogenic:** I.N.H
7. **Infection:** ☛ **Diphtheria:**{ mainly **motor** & bilateral affection & 10th cranial N. affection} MCQ
☛ **Leprosy:** {mainly **sensory** & loss of all sensations & 5th, 7th cranial Ns. affection} MCQ
ttt. 1-Dapson
2-Rifampicin
3-Cipa 1906
8. **Immune:** →Collagen disease
→**Guillan Barre \$**
Ascending paralysis & Cytoalbuminous dissociation
3- phases: **1** Viremia (FHMA)
2 Latent (Asymptomatic)
3 Paralytic {mainly motor & 7th, 10th cranial Ns. Affection} MCQ
ttt

☒ Plasma Phoresis	☒ Cortisone
☒ Immunoglobulin.	☒ O2 ventilation
☒ ttt. Of DM.	

Myasthenia Gravis (M.G.)

Definition:

It's a neuromuscular disorder characterized by easy fatigability of skeletal muscles, specially on repetition of movement.

Etiology:

Auto-immune disease in which there are **A.Choline receptors antibodies**

80% of cases associated with thymic dysfunction

50% of cases associated with thyrotoxicosis

Clinical Picture:

♀ > ♂ ☺

➔ Easy fatigability (**periodic skeletal muscle weakness**) characterized by:

- ① Diurnal variation with worsening of symptoms in the after noon
- ② Descending march
 - **Ocular manifestations:** Ptosis & Diplopia
 - **Jaw muscles:** mouth hanging open
 - **Bulbar manifestations:** dysarthria: Hoarseness of voice
 - **Neck muscles:** tilting of head
 - **Skeletal muscles of UL & LL:**
 - ☛ Proximal > distal : e.g ; Hair Combing.
 - ☛ UL > LL
- ③ No Sensory loss.
- ④ Associated thymic enlargement, hyperthyroidism.

Diagnosis:

1. Clinical test:

Induction of fatigue by repetition of movement :

- Forward arm abduction time
- Counting from 1 to 50 → dysarthria & nasal tone
- **Walker's test:** rise BP>Systolic, then Ask the patient to do rapid repetitive movement with his hand till exhaustion occur and then releasing the cuff
➔ Ptosis

2. Pharmacological test:

- **Prostigmine test:** (anticholinesterase)
Improvement of Ptosis within **20-30** minutes.
- **Tensilon (edrophonium) test:**
Improvement of Ptosis within **2** minutes.

3. Serological:

- Anti-acetylcholine Receptor (Anti-AchR) Antibodies
Is **+ve** in more than **80%** of cases

4. Muscle Biopsy & EMG.

5. X-ray chest to detect thymic enlargement

6. Thyroid function tests.

Treatment:

⇒ Medical

- ① **Prostigmine:** tab. 15mg & ampoule 0.5 mg
Onset: after 1/2hs. & duration : 3hs
- ② **Pyridostigmine:** tab. 60mg & ampoule 1mg
Onset: after 2 hs. & duration : 8hs

So, we can combine Prostigmine and Pyridostigmine and give them to the patient every 8 hours

- ③ **Tensilon (edrophonium):** IV. Only in myasthenic crisis
- ④ **Cortisone:** Prednisone 60mg/day then 10mg/day maintenance dose
- ⑤ **Immuno suppressive:** Azathioprine or Cyclophosphamide

⇒ Plasma phoresis

⇒ Surgical → Thymectomy.

Types of Myasthenia:

1. 1ry. Myasthenia: Discussed before
2. Neonatal Myasthenia:

- ✎ It occur in **20%** of babies born to myasthenic mother.
- ✎ Symptoms usually start within **3 days** of birth
- ✎ c/p. poor sucking & Poor crying.
- ✎ ttt. Prostigmine (0.05 -0.1mg) iv every 4 hours

3. Eaton-Lambert Myasthenic \$:

- ⇒ It may occur as paramalignant manifestations e.g **Small cell carcinoma**
- ⇒ It may also occur with other Auto-immune diseases
An IgG antibodies bind to nerve terminals → ↓↓ release of A.choline
- ⇒ Invest. EMG & X-ray chest for bronchogenic carcinoma
- ⇒ ttt. Pyridostegmine - Guanidine – cortisone

4. Myasthenic crisis:

- ⇒ paralysis of **respiratory** or **swallowing** muscles may occur.
- ⇒ ttt. Edratropium & plasma phoresis & Mechanical ventilation.

Wasting of small muscles of the hand

The small muscles of the hand are supplied by C₈ and T₁ spinal segment

Median nerve palsy usually due to carpal tunnel syndrome.	<i>Suggested by:</i> wasting of thenar eminence. Weakness of thumb flexion, abduction and opposition. Unable to lift thumb with palm upwards but able to press with index finger. Loss of sensation over palmar aspect of radial 3.5 fingers of hand. <i>Confirmed by :</i> nerve conduction study results.
Ulnar nerve lesion	<i>Suggested by:</i> wasting of hypothenar. Able to lift thumb with palm upwards but unable to press with index finger. Weakness of finger abduction and adduction. Loss of sensation in ulnar aspect 1.5 fingers of hand. <i>Confirmed by:</i> nerve conduction study results.
T1 lesion: anterior horn cell or root lesion	<i>Suggested by:</i> wasting of all small muscles of hand. Unable to lift thumb with palm upwards , unable to press with index finger. <i>Confirmed by:</i> nerve conduction study results. MRI scan appearance around T1 level.
Motor neurone disease	<i>Suggested by:</i> <u>signs of T1 lesion</u>, prominent fasciculation, spastic paraparesis, wasted fasciculating tongue, <u>no sensory signs</u>. <i>Confirmed by:</i> absence of structural abnormality on MRI scan appearance.
Syringomyelia	<i>Suggested by:</i> <u>signs of T1 lesion</u>, fasciculation not prominent, burn scars, dissociated sensory loss, Horner's syndrome, nystagmus. History over months to years. <i>Confirmed by:</i> MRI scan appearances.

Neurology

In Capsule Series

Any prolonged systemic illness	<i>Suggested by:</i> global muscle wasting, general weight loss. <i>Confirmed by:</i> improvement in muscle wasting if primary disease treatable.
Cervical spondylosis	<i>Suggested by:</i> signs of T1 lesion , neck pain and stiffness, and referred pain. <i>Confirmed by:</i> MRI
Tumor compressing nerve root	<i>Suggested by:</i> signs of T1 lesion , referred pain. Progressing over months. <i>Confirmed by:</i> MRI
Brachial plexus lesion	<i>Suggested by:</i> signs of T1 lesion and history of trauma to shoulder area or birth injury. <i>Confirmed by:</i> nerve conduction study results.
Cervical rib	<i>Suggested by:</i> signs of T1 lesion aggravated by movement or posture. <i>Confirmed by:</i> X-ray . Relief by surgery.
Pancoast tumour	<i>Suggested by:</i> signs of T1 lesion , Horner's syndrome, features of lung cancer (clubbing, chest signs, lymph nodes etc.). <i>Confirmed by:</i> CXR and CT scan appearances.

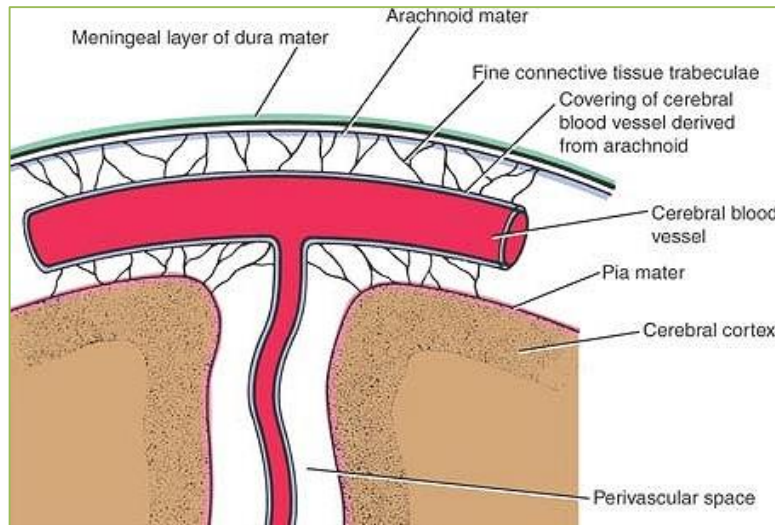
*A Physician without differential diagnosis is like a
Soldier without his gun, like a Clown without his fun.*

AL-SAYED DAWOUD

Subarachnoid hemorrhage

Definition :

- Sudden arterial bleeding into the subarachnoid space.
- Around 5% of all strokes are due to SAH.

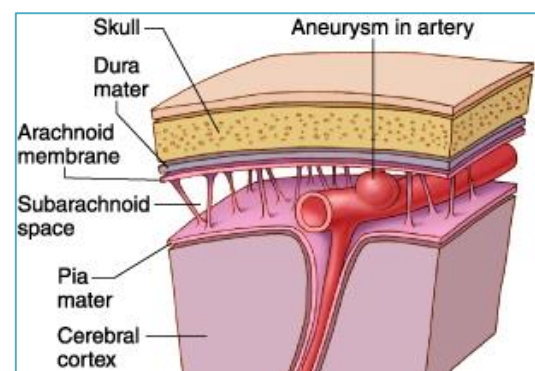


- There are three layers of meninges, known as the dura mater, arachnoid mater and pia mater.
- Just remember **I PAD** (Pia, Arachnoid, Dura) from deep to superficial.
- The subarachnoid space is the space that normally exists between the arachnoid and the pia mater, which is filled with cerebrospinal fluid.

Etiology :

1. Ruptured **intracranial aneurysm** (**Congenital**, mycotic) :

- Most common cause of spontaneous (Non traumatic) SAH. 80%
- Multiple aneurysms may occur in families or with systemic diseases such as polycystic kidney, Marfan syndrome, Ehlers-Danlos syndrome and coarctation of aorta.

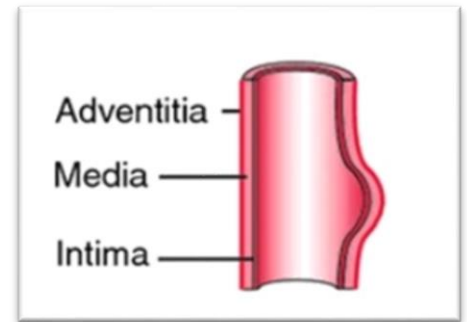


2. Angiomatous malformation : 10%

3. Head trauma : In general, head trauma is the most common cause, but traumatic subarachnoid hemorrhage is usually considered a separate disorder.

Pathophysiology :

- Most aneurysms (85%) occur in anterior circulation, mostly on the **circle of Willis** e.g.
 - Junction of the anterior communicating and anterior cerebral arteries.
 - Junction of the posterior communicating and internal carotid arteries.
 - Bifurcation of middle cerebral artery.
- These berry aneurysms develop gradually over the lifetime of the patient, arterial internal elastic lamina disappears & media thins. Connective tissue replaces smooth muscle cells. They are usually silent until 40-50 years of age. The risk of rupture increases as the size of the aneurysm increases. Aneurysms larger than 10 mm in diameter are five times more likely to rupture than are smaller ones.
- At rupture, blood in the subarachnoid space causes a chemical meningitis and increases intracranial pressure for days or weeks.
- Secondary vasospasm may cause focal brain ischemia.
- A 2nd rupture (rebleeding) sometimes occurs, most often within 2 weeks. ☠☠

**Clinical picture :**

- **Incidence** : 9 per 100,000 per year. Higher in Finland and Japan. 😓
 - **Sex** : the incidence of SAH is 1.6 times higher in women than in men.
 - **Historical red flags** : positive family history, polycystic kidney disease, uncontrolled hypertension.
 - **Age** :
 - Rupture of aneurysm : usually 40-50 years.
 - Rupture of arteriovenous malformation : usually 20-30 years.
- 1) **Pre rupture** : **2T**, Most aneurysms remain **asymptomatic** until they rupture
- A. **T**ension Headache.
 - B. **T**ransient focal lesion :
 - Compression on cranial nerves esp. 2,3,4,5,6 nerves
 - Aphasia, hemiplegia, ...

2) At rupture :

- Risk factors : smoking, excessive alcohol intake and hypertension.
- Clinical manifestations are classified into **3 groups :**

1. Manifestations of rapidly increasing intracranial tension :

- a) Headache : **Sudden**, Severe & splitting *“The worst-ever headache”*
- b) Nausea & vomiting often occur.
- c) Drowsiness or coma may follow immediately & continue for hours to days.
- d) Bradycardia and irregular respiration.
- e) Papilledema : may be present and may be accompanied by retinal and vitreous hemorrhage.

Suspect subarachnoid hemorrhage if headache is severe at onset and reaches peak intensity within seconds or causes loss of consciousness.

2. Manifestations of meningeal irritation :

after 3 - 12 H



- a) Neck rigidity on passive flexion.
- b) **Kernig's sign** : Extending the knee with the patient lying supine with the hip & knee joints flexed at 90° ⇒ stretches the nerve roots and cause meningeal pain.
- c) **Brudzinski's sign** :
 - **Neck sign** : passive flexion of the neck leads to flexion of both knees and hips.
 - **Leg sign** : passive flexion of one hip (while the knee is extended) leads to flexion of the other hip and knee.
- d) Pain in the back and lower limbs : due to irritation of cauda equine.
- e) Fever.

3. Focal manifestations :

- a) Cranial nerve palsies (usually ocular) : due to compression of hematoma.
- b) Cerebral infarction (e.g. Hemiplegia, aphasia) : due to compression of hematoma & spasm of cerebral arteries.

Complications :

I. Neurological complications : 📌

1. Aneurysm **rebleeding** : occurs in 20% of patients within 2 weeks. After 6 month, a second rupture occurs at a rate of 3% per year. Approximately 80% will die.
2. Cerebral **vasospasm** : occurs in 40% of patients usually in the first 2 weeks.
3. Hydrocephalus.

II. Non-neurological complications : 📌

1. Stress hyperglycemia.
2. Myocardial infarction.
3. **Hyponatremia** : It is the most common electrolyte abnormality seen in patients with aneurysmal SAH. It is due to 2 mechanisms, syndrome of inappropriate anti-diuretic hormone (SIADH) and cerebral salt waste syndrome.

Investigations : 📌

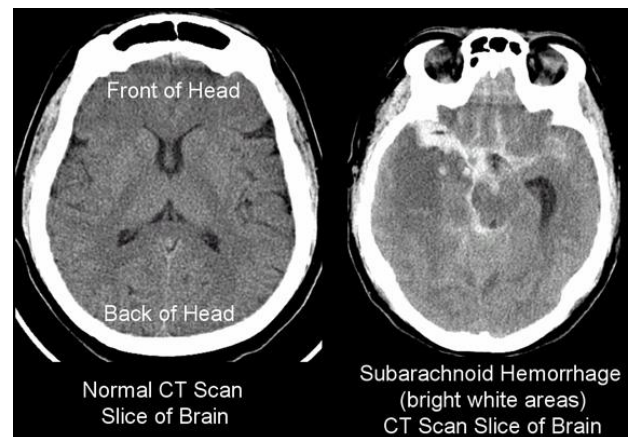
1. **CT scanning** : is the **first choice** & usually shows subarachnoid blood.

2. Lumbar puncture & CSF examination :

(if CT scan is normal or not available)

- a) Bloody CSF : a traumatic tap must be keep in mind.
- b) Xanthochromia (yellow color of the CSF) : due to RBC lysis.
- c) High pressure.
- d) Slight lymphocytosis.
- e) Blood clotting : doesn't occur.

3. **Angiography** : to localize aneurysms & angiomatous malformations. It is delayed in patients with a poor clinical condition.



Grading : “Hunt and Hess Severity Scale”

Grade	Criteria
1	Asymptomatic, mild headache.
2	Moderate to severe headache, neck rigidity, no focal deficit other than cranial nerve palsy
3	Mild mental status change (drowsy or confused), mild focal neurologic deficit.
4	Stupor or moderate to severe hemiparesis.
5	Deep coma, decerebrate rigidity (extensor posturing)

**Treatment :****I. General management of coma :**

- a) Absolute bed rest & balanced nutrition.
- b) Avoid straining (stool softener & antitussive drugs)

II. Symptomatic treatment :

a) Analgesia :

- Paracetamol.
 - Morphine.
- with less side effects. (strong analgesics may depress conscious level and mask deterioration)

Avoid aspirin, bleeding baby

b) Antiemetic Agents.

c) Sedative : Benzodiazepine (midazolam) may be given.

d) Anticonvulsants (Phenytoin) : in patients with seizures.

e) Cerebral dehydrating measure : mannitol & lasix to lower ICP and cerebral edema.

f) Symptomatic cases of hydrocephalus may be managed by lumbar puncture.

III. Control of hypertension :

- With nicardipine or beta blockers.
- Optimally, systolic blood pressure of no more than 130-140 mmHg should be the goal, unless clinical evidence of vasospasm is noted.

IV. Nimodipine (Ca channel blocker) : 60mg orally/4h for 3 weeks to reduce vasospasm.

- The use of aminocaproic acid for hemostasis is controversial.
- Needless to say that anticoagulants and antiplatelet drugs are contraindicated.

V. Surgical / Interventional treatment : (Clipping or coiling of aneurysms)

Once the patient is stabilized, angiography is done to localize the cause then one of the following :

a) **Surgery :** Clipping of the aneurysm. Figure 1

b) **Endovascular coiling** (*Embolization to close the aneurism*) : Figure 2

Placing platinum coils within the aneurysm via a catheter that is passed from the femoral artery. The aneurysm is packed tightly to enhance thrombosis and over time is walled-off from the circulation.

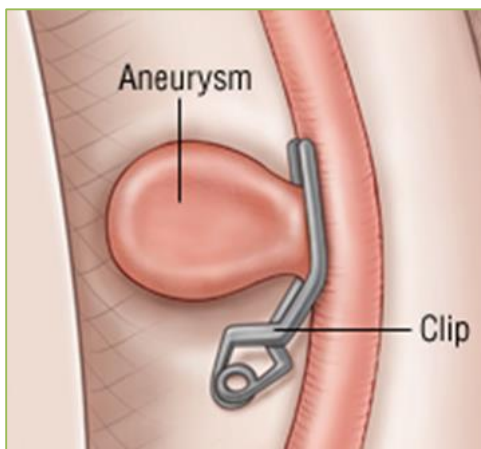


Figure 1

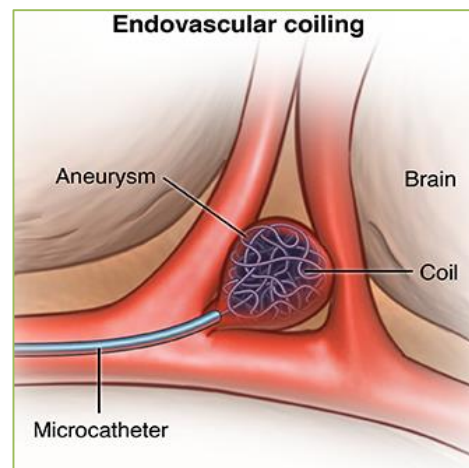


Figure 2

Surgical clipping or endovascular coiling of the ruptured aneurysm should be performed as early as possible to reduce the rate of rebleeding.

Prognosis :

- 15% die before reaching the hospital.
- About 25% die after the first rupture within 24 hours.
- Another 15% die within few weeks due to rebleeding.
- After 6 month, a second rupture occurs at a rate of 3% per year. 80% will die
- More than one third of survivors have neurologic and cognitive deficits.
- Prognosis depends on the age, quantity of subarachnoid blood on CT, level of consciousness, & presence of hypertension.

Ataxia

Definition: Incoordination of voluntary movements with or without disequilibrium
In absence of paralysis or paresis.

Types:

(1) Cerebellar Ataxia

Etiology:

1. Heridofamilial → Friedrich's ataxia & Marie's ataxia
2. Vascular → PICA & AICA
3. Toxic → Alcohol & Barbiturates
4. Neoplastic → Medulloblastoma

Clinical picture:



☞ Ataxia

1. Limb:

a. kinetic tremors and dysmetria , detected by:

- ⇒ Finger to nose test
- ⇒ Finger to finger test
- ⇒ Heel to knee test
- ⇒ Buttoning and unbuttoning

b. Adiadokinesia

c. Rebound phenomenon

2. Eye: Nystagmus

3. Tongue: Stacatto Speech

☞ **Hypotonia** marked with acute lesion

☞ **Disequilibrium** → Wide Gate
→ Drunken Gate
→ Zigzag Gate (if bilateral)

	Friedrich's ataxia	Marie's ataxia
Degeneration of :	Cerebellum and 3p ☞ Pyramidal tract ☞ Peripheral nerves ☞ Posterior column	Cerebellum and Pyramidal tract
Features:	+ve. Babiniski Sign Hypotonia & Hyporeflexia	+ve. Babiniski Sign Hypertonia & Hyperreflexia
Association:	Skeletal deformities Cardiomyopathy	Mental Retardation Optic neuritis

(2) Sensory ataxia

Definition: Ataxia due to loss of deep sensations

Etiology:

1. Peripheral Nerves: Peripheral neuropathy
2. Posterior column: SCD
3. Posterior root: Tabes dorsalis
4. Thalamus : Thalamic syndrome
5. Cortical sensory area :Parital lobe lesion

Clinical picture:

- ☞ Clinical picture of ataxia (*on eye closure*)
- ☞ Loss of deep sensation: loss of vibration sense & loss of position and joint sense
- ☞ Stamping gait
- ☞ Romberg's Sign (**Inability to maintain the upright position with eye closed**)

(3) Vestibular ataxia

Definition: Lesion of vestibular division of 8th cr.nerves

Etiology: Menier's disease & labrynthitis

Clinical picture:

- ☞ Vertigo & Tinnitus
- ☞ Nustagmus & disequilibrium.

Extra Pyramidal System

Definition:

Any fibers that can influence movement **Without** passing in the pyramidal system.

Anatomy: see the figure

Function: 

1. Filtration of motor impulses from area 4
2. Regulation of muscle tone
 - ☞ Acetyl Choline → Activate (↑ Muscle tone)
 - ☞ Dopamine → Decrease (↓ Muscle tone)
3. Emotional, associative and automatic movements.

So, any lesion in the extra pyramidal System is characterized by: 

① Involuntary movement: (8 characters)

- ⇒ Involuntary & static & ↑↑ by stress & ↓↓ with sleep.
- ⇒ In parkinsonism: (Regular & Rhythmic & Distal > Proximal & Money count)
- ⇒ In Chorea : (irregular & dysrhythmic & proximal & purposeless)

② Muscle tone : all increase muscle tone Except chorea

③ Emotions:

- ⇒ Parkinsonism → bradykinesia
- ⇒ Chorea → exaggerated (emotional instability)

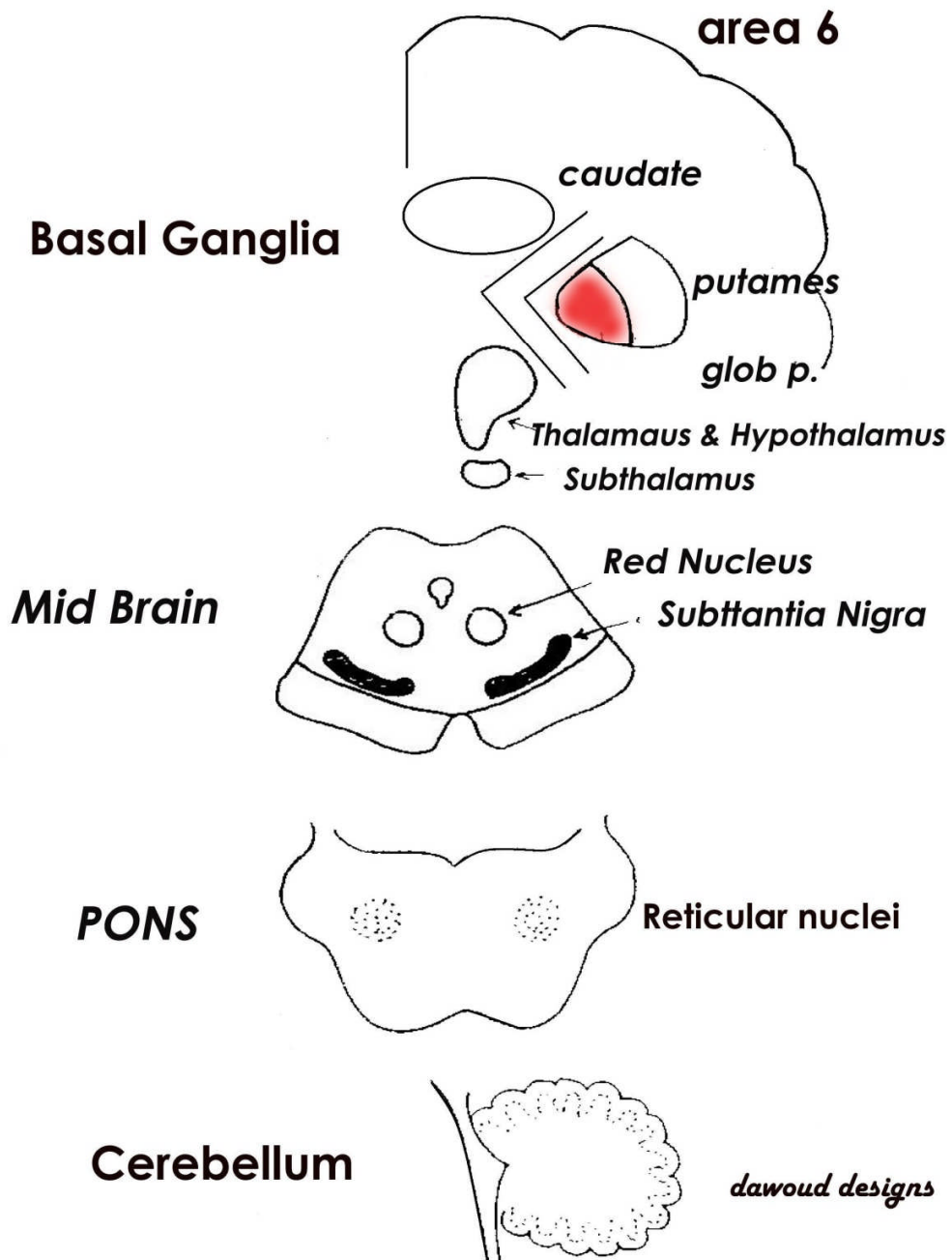
Addiction-Free Nation Program should consider **in capsule series** its 1st priority, as it has been proved that almost all internal medicine seekers are addicted to it.

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Parkinsonism

Neurochemical basis:

↑ Dopamine in the basal ganglia and substantia nigra

↓ Acetyl Choline

Etiology

1. Idiopathic (paralysis agitans) → *The commonest type*
☞ Degenerative type of the disease
2. Post Encephalitis
3. Cerebral Atherosclerosis

others:

4. Wilson's disease
5. Toxic : Co & Mg
6. Tumor
7. Trauma : Rare

Clinical Picture: *B.Ri.T.ch man*

Triad of:

- 1-Tremors
- 2-Rigidity
- 3-Bradylalia

1-Tremors:

4

- Involuntary
- Static
- ↑↑ by Stress
- ↓↓ by Sleep

8 (hyperkinesia)

4

- Rhythmic
- Regular
- Distal > Proximal
- Money count(4-8/sec.)

☞ D.D. of Tremors:

- | | |
|----------------------|--------------------------|
| 1) Parkinsonism | 5) Cerebellar tremor |
| 2) Senile tremor | 6) Thyrotoxicosis |
| 3) Familial tremor | 7) Flapping tremor |
| 4) Hysterical tremor | 8) Alcohol & Amphetamine |

2-Rigidity:

	Rigidity	Spasticity
1-Site of lesion 2-Distribution 3-Charactres 4-Deep reflexes	-Extra pyramidal tract -proximal > distal -flexors of UL&LL -lead pipe or cogwheel -heporeflexia	-Pyramidal tract -distal > prximal -flexors of UL& extensors of LL -clasp kife. -hyper reflexia

⚡ D.D. of Hypertonia

- 1-Pyramidal lesions (UMNL): Claps knife type
- 2-Extra pyramidal lesion : Parkinsonism (Lead pipe or Cog weel)
- 3-Hysterical
- 4-Toxic: Resirpine , Co : Cog weel rigidity.
- 5- Meningitis
- 6-Myotonia

3-Brady Kinesia:

- 1-Monotonus speech
- 2-Mask face
- 3-Lack of blinking
- 4-No swinging of arm

**** Difference between the most common 3 causes of Parkinsonism**

	diopathic	Encephalitis	Thrombosis
1-Age 2-Onset 3-Course 4-Tremors & rigidity 5-Association	-Around 50 -Gradual -Progressive -Tremors> rigidity - NONE	- <40 -Acute -Regressive -Rigidity > tremors -Occulogyric Crisis -Pyramidal signs: * Scuating * Sialorrhea * D.I. * Obesity * Impotence -History of Encephalitis may be obtained	- 40-60 years -Gradual -Remission &exacerbation -Rigidity> tremors -Hypertension -D.M. -Coronary H.D. -Pyramidal manifestations

Investigations: NO specific investigations ☺

-Routine investigations (MRI & CT& Tests for Wilson's D.
e.g. caeruloplasmin and serum cooper)

Treatment:**1. Anticholinergic drugs**

➔ benztropin (Congentin) & tremaril

Side effects :

- ✗ Retention of urine .
- ✗ Tachycardia.
- ✗ Dryness of mouth.

2. L – dopa

➔ It is transformed to dopamine peripherally which can't pass BBB.

Side effects :

- ✗ psychosis
- ✗ palpitation
- ✗ GIT upset
- ✗ Chorea → *On and Off phenomenon*

3. Sinemet

➔ (L.dopa + Carbidopa) inhibit the peripheral decarboxylation of L-dopa into dopamine So L – dopa will pass through BBB.

4. Amantadin

➔ uptake of dopamine by the neurons and used as adjuvant therapy.

5. Ms relaxant and B.B

Computerized by: Dr/Alsayed Dawoud

Dr/Ismail Ezzat

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Chorea

Definition:

Involuntary,static,irregular,dysrhythmic,sudden,jerky,pseudopurposive movement of any part of the body due to lesion in caudate nucleus.

♀ > ♂ ☺ (↑ dopamine - ↓ Ac.choline)

Etiology:

1. **Idiopathic** (*Senile chorea*)
2. **Encephalitis** (*post encephalitic chorea*)
3. **Vascular** (*Hemiballismus*)
4. **Toxic** (*chorea gravidarum*)
5. **Heridofamilial** (*Huntington's chorea*)
6. **Autoimmune** (*Rheumatic chorea*)

Rheumatic chorea {Sydenham's chorea}

c/p:

I. Movement:

- ↪ 4 characters **as** Parkinsonism
{Involuntary & static & ↑↑ by stress & ↓↓ with sleep}
- ↪ 4 characters **opposite** Parkinsonism
{Irregular & dysrhythmic & proximal & purposeless}

II. Hypotonia:

- ↪ Dancing gait
- ↪ Grimacing smile
- ↪ No tongue protrusion without teeth support
- ↪ Arm deviation down when elevated
- ↪ Scaphoid hand → flexion at the wrist
→ Over extension of metacarpophalangeal and interphalangeal joints

III. ↑ Emotions:

Emotionally instability as sudden crying or laughter

Clinical varieties of Rh. Chorea

- ⇒ Chorea **gravis** (*disorder of Sleep, Speech & Swallowing*)
- ⇒ Chorea **mollis** (*flaccid paralysis*)
- ⇒ Chorea **mania** (*instable emotions*)

Treatment:

☑ ttt. Of Rheumatic fever

- ✖ Complete bed rest
- ✖ Acetyl salicylic acid 6-8mg/day
- ✖ Prednisone in rheumatic activity

☑ Dopamine antagonists

- ✖ Hallopridol & Reserpine

☠ Huntington's chorea ☠

- ✖ Autosomal dominant → chromosome **4** MCQ
- ✖ Middle age
- ✖ Gradual onset & Progressive course → Fatal type of chorea
- ✖ Due to degeneration of: #→ **Basal ganglia**

{Gross movement with interference with feeding and walking}

#→ **Cerebral cortex (prefrontal area)**

{Change in behavior and mentality}→ Dementia is prominent

☠ Hemiballismus chorea ☠

- ✖ It is a form of forceful, flinging, high-amplitude, coarse, chorea.
- ✖ Involuntary movements usually affect only one side of the body
- ✖ Due to lesion involving the contralateral **subthalamic nucleus (STN)** MCQ
- ✖ Subside spontaneously within several weeks

Athetosis

1) Involuntary movements: (8 criteria)

- ⇒ Involuntary & Static & ↑ by Stress & ↓ by Sleep
- ⇒ Irregular
- ⇒ Dysrhythmic
- ⇒ Distal > Proximal
- ⇒ Snake like movement

4

2) Hypertonia: *during the movement*

3) Facial grimacing & dysarthria may occur

Dystonia

1) Involuntary movements

- ⇒ Involuntary & Static & ↑ by Stress & ↓ by Sleep
- ⇒ Very Slow twisting like movements
- ⇒ Affects neck & trunk & proximal extremities

2) hypertonia : *during the movement*

3) No emotional manifestations MCQ

- ⇒ -ve reflexes & Normal sensations, mentality & Speech

🔊 DD. of involuntary movements: ☠

1. Epilepsy → Convulsions
2. Extra pyramidal lesion → Parkinsonism & Chorea & Athetosis & Dystonia
3. LMNL → Irritative lesion
4. Psychomotor movements → Tics
5. Tremors

Motor Tics

Involuntary, Abrupt, Sudden, Isolated, Brief movements

Types:

Simple: e.g → Eye blinking

Complex: e.g → kicking & pelvic gyration

Headache

Definition: Pain over the level of the eye brows to the sub-occipital area.

Sensitive structures in the skull

1. All the tissue covering the bone except the hair
2. Cerebral arteries
3. Venous sinuses
4. Cranial nerves: 5,9,10 & upper 3 cervical nerves
5. Meninges: especially the basal

N.B: *the Brain itself is insensitive*

Etiology

1) Vascular headache : (V.D.)

- ⇒ Migraine
- ⇒ Hypertension & Hypoxia & Hypoglycemia
- ⇒ Arteritis & Aneurysm
- ⇒ Toxic: → Caffeine withdrawal & Alcohol
→ renal and hepatic failure

2) Traction Headache: (*Space occupying lesion*)

- ⇒ due to stretch of Meninges as in brain tumors

3) Tension Headache: (*Psychogenic headache*)

- ⇒ Cap-like constriction (Ache pain)
- ⇒ Not localized & Not throbbing & Don't increase with Straining
- ⇒ Usually associated with depression, neurosis & stress

4) Meningeal irritation:

- ⇒ Meningitis
- ⇒ Subarachnoid hemorrhage

5) Muscle contraction headache:

- ⇒ Prolonged contraction of the muscles of the head and neck e.g: prolonged driving

6) Neuritis and neuralgia:

- ⇒ Of the sensory nerves of the scalp e.g.: trigeminal neuralgia

7) Referred headache: *may be very sever*

- ⇒ Eye: **iritis, glaucoma**
- ⇒ Ear: **middle ear diseases**
- ⇒ Nasal sinuses diseases
- ⇒ Teeth diseases and Tongue diseases

Criteria of a Serious Headache ?

↑ ICT ←

le Series

- 1- Sudden, Severe Headache
- 2- Headache associated with projectile vomiting and blurring of vision
- 3- Headache that begins after an injury of the head
- 4- Headache followed by ~~ICL~~ or associated body weakness
- 5- Headache associated with fever and a stiff neck.

Neurology

Headache

Migraine

Definition:

Paroxysmal attacks of throbbing headache usually unilateral rarely preceded by aura.

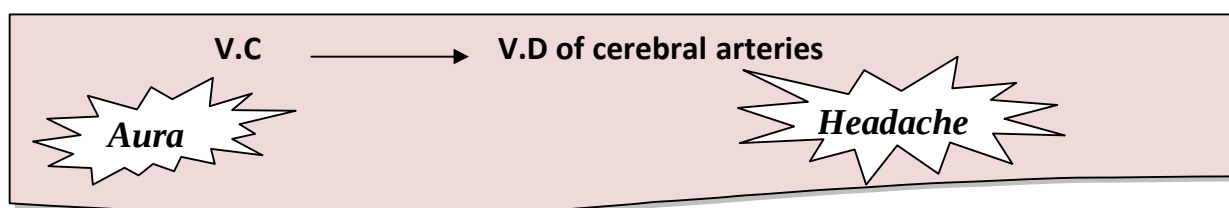
Etiology: the exact cause is unknown

⇒ ♀ > ♂ ☺

⇒ Genetic factor may play a role 70% Heridofamilial MCQ

⇒ Type A personality

Mechanism: ischemia followed by V.D



Theories:

- ✂ **Serotonin theory:** initial increase in serotonin → V.C of cerebral arteries
Then, degeneration of serotonin → V.D of cerebral arteries
☞ Serotonin receptors in the other parts of the body are affected {nausea & vomiting}
- ✂ **Hypoxic theory:** V.C of cerebral arteries followed by reactive V.D

Clinical picture: (4 stages)

① **Prodroma:** changes in mood or appetite days before the attack.

② **Aura:** immediately before the attack

2 V (Visual hallucination & Vertigo)

2 P (Parasthesia & paresis for about 5 minutes)

③ **Headache:**

1. **Site:** usually unilateral (starts in the temple or around the eye)
2. **Type:** throbbing pain (*very sever*)
3. **Duration:** lasting 4 to 72 hours
4. **Increased by:** exercise, light & menstruation
5. **Decreased by:** sleep
6. **Association:** nausea and vomiting

④ **Post headache phase:** polyuria

Types of migraine:

1. **Classic migraine:** migraine with aura 15% only MCQ
2. **Common migraine:** without aura 85%
3. **Variants:** according to aura {Hemiplegic or Ophthalmoplegic migraine }
4. **Cluster headache :** Horton's syndrome = **Migranous Neuralgia**

 Migranous Neuralgia

- *Frequent attacks of headache which is usually associated with conjunctival injection, lacrimation, rhinorrhea, flushing and sweating of the affected side of the face.*
- ♂ > ♀ middle age
- ↑ *By histamine, alcohol or heat*
- *ttt.O2 inhalation 100% & Ergotamine*

Treatment:**☑ During the attack (4)**

1. Rest in quite room
2. Analgesics: aspirin or paracetamol in mild attack
3. **Ergotamine** (serotonin agonist)
 - ☞ Oral, sublingual or nasal spray
 - ☞ Migranil® or Imigran®
4. Antiemetics

☑ Between the attack (4) A,B,C,D

1. Avoid precipitating factors
2. **B**.Blockers: prevent V.D
3. CCB: flunarizine HCL
4. **Deseril:** serotonin antagonist

↪ **S.E:** retroperitoneal fibrosis MCQ

Status Migrainosus

Def.: migraine attacks that persist for longer than 72 hours despite treatment

C/p: ⇨ headache-free periods for less than 4 hours may occur

⇨ **Usually associated with prolonged use of analgesics**

TTT. → **Hospitalization**

→ **ttt with detoxification**

Disorders of skeletal muscles

Myopathies

Definition :

- Myopathies are disorders in which there is a **primary** impairment of skeletal muscles.
- Weakness of muscles is a characteristic finding in myopathies. Each muscle disease has a particular pattern of involvement.

Classification of myopathies :

I. Hereditary myopathies : "Progressive muscular dystrophy"

1. X-linked recessive : affect only males

- Duchenne muscular dystrophy.
- Becker muscular dystrophy.

2. Autosomal recessive :

- Limb girdle (usually scapulo-humeral)
- Congenital muscular dystrophy

3. Autosomal dominant : the patterns of weakness are described in their names, *baby*

- Facio-scapulo-humeral dystrophy.
- Scapulo- peroneal dystrophy.
- Distal : onset at 40 – 60 years old.
- Ocular : bilateral symmetrical affection, bilateral ptosis & external ophthalmoplegia.
- Oculopharyngeal : onset at 40 years old. Similar to ocular type but with dysphagia.

II. Acquired myopathies :

1. Inflammatory myopathies : Dermatomyositis, polymyositis.
2. Endocrine myopathies : Hyper- or hypo adrenal disorders, Hyper- or hypothyroidism, hyper- or hypoparathyroidism & acromegaly.

3. Metabolic myopathy : glucose metabolism disorders, lipid metabolism disorders, mitochondrial disorders.

4. Toxic myopathy :

- Alcohol.
- Penicillamine.
- Cortisone, Colchicine, Cholesterol lowering agents (Statins).

Hydrophilic statins (rosuvastatin) are less likely to cause myopathy than lipophilic statins (atorva, simva, lova-statins)

Pathogenesis :

- A defect in muscle fiber plasma membrane is leading to uncontrolled entry of Ca which activates Ca dependent protease that induces muscle fiber necrosis.
- The missing gene protein is **dystrophin**.

Clinical picture :

General characteristics of progressive muscular dystrophy : just 13 items 🤔

1. It usually affects the first & second decades.
2. Positive family history.
3. Bilateral & symmetrical.
4. **Proximal** than distal. It usually affects the muscles which develop early during intrauterine life (pelvic girdle, shoulder girdle & trunk).
5. The weakness is of LMNL : hypotonia, hyporeflexia.
6. Winging of scapula : due to weakness of serratus anterior & trapezius.
7. Exaggerated lumbar lordosis : due to weakness of extensors of trunk.
8. Pot belly abdomen : due to weakness of abdominal muscles.
9. Waddling gait : due to weakness of abductors of the hip (gluteus medius & minimus)
10. Gower's sign "climbing test" : characteristic manner of getting up from the floor, he climbs himself due to weakness of gluteus maximus.
11. **No** Sensory changes.
12. **No** bladder disturbance.
13. Sternomastoid & clavicular head of pectoralis major are **NOT** involved.



Causes of myopathy with peripheral neuropathy :

- **A**lcohol.
- **P**araneoplastic syndrome.
- **C**onnective tissue diseases.
- **D**M
- **E**ndocrinal disorders.

Duchenne muscular dystrophy :

- A. It is an X linked recessive disorder caused by an absence of dystrophin occurring in 1/3000 male infants.
- B. It affects pelvic girdle then shoulder girdle muscles.
- C. Gradual with rapidly progressive course : there is no abnormality at birth, but the condition (slow walking, inability to run & frequent falling) is apparent by the 4th year. The boy is usually wheelchair bound by 10 years old, and death is usually by the age of 20, from **respiratory or heart failure**.
- D. There is **pseudohypertrophy** of the calves, quadriceps & glutei muscles. Needless to say plus all general criteria of muscular dystrophy.

Becker's muscular dystrophy :

- It is also an X linked recessive disorder but it is slowly progressive and not shorten life. Dystrophin is altered rather than absent. Cramps associated with exercise are common.

	Duchenne	Becker
– Age of onset	1 st decade	2 nd & 3 rd decade
– Course	Rapidly progressive	Slowly progressive
– Skeletal deformities	Present	Absent
– ECG changes	Present	Absent

Investigations : The diagnosis of myopathy is often made clinically

1. **Urine :** Appearance of **Creatine** & diminished of creatinine, because the diseased muscles can't metabolize creatine to creatinine.
2. **Serum enzymes :** CK (Creatine phosphoKinase) is highly elevated ($> 10,000\text{U/L}$). CK is often raised in carrier females although there are no clinical manifestations.
3. **EMG (ElectroMyoGraphy) :** Diminished duration & amplitude of motor action potential.
4. **Muscle biopsy :**
 - **Swelling** of some muscle fibers.
 - **Striation** is less marked
 - **Splitting** of muscle fibers.
 - **Staining** for dystrophin is absent.
 - **Fatty infiltration** & **Fibrosis**.

Treatment : There is no cure for myopathy, just **supportive** treatment

- Steroids may produce short term improvement (risk exceeds benefit).
- Myoblast injection into the muscle (cost exceeds benefit).
- Ca antagonists.
- Physiotherapy.
- Psychological treatment.
- Genetic counselling is important. Prenatal diagnosis is available.

Brain tumors V.I.

Def: Space-occupying neoplasms in the cranial cavity.

Classification:

I-1ry Tumors

- From the meninges → Meningiomas
- " " brain tissue → Gliomas
- " " pituitary → pituitary Tumors.
- " " cranial nerves → Acoustic Neuroma of 8th nerve.
- " " blood vessels → Hemangiomas Hemangioblastoma.

II- 2ry Tumors: Metastasis.

Pathophysiology:

- 1) Damage of Specific Neural pathways traversing the brain.
- 2) infiltration of normal parenchymal tissue, disrupting normal function.
- 3) ↑ Ict Through:
 - Space occupying lesion
 - Neovascularization → Brain edema.
 - Breakdown of TEJ → x BBB.
 - obstruction of CSF flow (obstructive hydrocephalus).
 - Brain injury → cytokine → ↑ permeability.

C/P:

1- Manifestations of ↑ Ict

1- Headache

- Dull aching
- Severe in the morning and tends to ↓
- ↑ by coughing, sneezing and straining.
- No significant value in Localization of the Site of the Tumour.

2- Vomiting (Due to ++ of vomiting center) projectile More frequent in the morning.

3- papilledema: (reliable sign of ↑ Ict).

- Blurring of vision occurs followed by gradual diminution of vision.
- Failure of vision occurs due to post-papilledemic optic atrophy.
- ophthalmoscopically
 - Blurring of margins of optic disc
 - Retinal Hge and exudates
 - optic atrophy.

4- others:

- Syncope
- DCL
- Cranial nerves (6th nerve palsy)
- Cushing triad (Bradycardia, HTN, Resp. depression).
- Herniation Syndromes:

a) Subfalcine

b) Transtentorial (uncal)

c) Tonsillar Herniation

2 Focal Manifestations

- 1- Frontal Lobe Tumors
- 2- Parietal Lobe Tumors
- 3- Temporal Lobe Tumors
- 4- occipital Lobe Tumors.
- 5- Cerebello-pontine angle Tumors (Acoustic Neuromas).

• Ipsilateral ataxia

- " affection of 5th, 7th, 8th cranial nerves.
- Cont. hemiplegia (pyramidal compression in the pons).

6- pituitary Tumors

A) supra sellar:

- 1- Hypothalamic
- 2- panhypopituitarism
- 3- Bitemporal Hemianopia.

B) Intraseellar.

- 1- Hormonal Manif: chromophobe ad.
- Acidophi ad → gigantism
- Basophil ad → Cushing's
- 2- Headache

- 3- Neurological manif:
 - 1- Ant. compression
 - 2- post. compression
 - 3- Lateral compression
 - 4- Superior compression.

Brain Tumours.

Neurological Manifestations.

- 1- Anterior compression :
 - optic chiasma : Bitemporal Hemianopia
 - optic Nerve : optic atrophy.
 - olfactory tract : Anosmia.
2. post. compression :
 - upper brain stem : Bilateral Δ Signs, ophthalmoplegia.
3. Lat. Compression :
 - optic tract = Homonymous hemianopia
 - Cavernous Sinus = cranial nerve (3, 4, 6, ophthalmic division of 5)
- 4- Sup. Compression :
 - Hypothalamus : Hypothalamic $\$$.

Inv:

- 1- MRI, CT scan
Best, most accurate
- 2- plain X-ray of skull
 - a) Signs of $\uparrow$ ICT (3S)
 - Silver-beaten appearance (finger prints)
 - Separation of cranial sutures
 - Sellar changes (in pituitary Tumors) (Sella Turcica)
 - b) Signs denoting the site of Tumor:
 1. Localised calcification.
 2. Localised Erosion.

3. Cerebral Angiography .
 - Abnormal vessels feeding tumor.
- 4- Air or myodil ventriculography
 - Filling defect
 - Displacement of Ventricular System.
5. ophthalmoscopic examination :
Papilloedema.

Treatment:

Surgical Removal of the Tumour,
(if possible)

Multiple Sclerosis

Definition:

This is a disease of unknown cause characterized by affection of white matter of the central Nervous system resulting in degeneration of its myelin sheath.

Etiology:

- ✎ Autoimmune: as CSF show ↑ immunoglobulins
- ✎ virus (*controversy*)

Clinical picture:

⇒ age → 20- 50 years

⇒ ♀ : ♂ → 3: 2

⇒ Onset → acute or gradual

⇒ Course → Remission & exacerbation

Mentality: Euphoric - emotional instability

Speech: Slurred , Staccato or Scanned

Cr. Nerve:

1. Optic → **Optic neuritis**
2. Facial → **LMNL & UMNL**
3. Oculomotor → **Diplopia & Ophthalmoplegia**

Motor: *usually UMNL* MCQ☑

1. paraplegic * Quadriplegic
2. monoplegic * hemiplegic

Sensory: Superficial & deep sensory loss ☞ +ve L'hermite's sign☞

Cerebellar: tremors & Nystagmus

Sphincteric disturbance & Impotence☠☺

Investigations:

- I. CSF Examinations: Gammaglobulin IgG & Oligoclonal bands
- II. Visual evoked potential to detect early affection of Optic Nerve.
- III. MRI: more sensitive during exacerbation
- IV. C.T. Scan → hypodense area in acute stage

Treatment of multiple sclerosis:

- * Treatment of acute exacerbations:
 - ☛ **Pulse steroid** therapy by **methyl prednisolone** 1 gm/D for 3 to 5 days.
- * Prevention of relapse:
 - ☛ Interferone.
 - ☛ Azathioprine.
- * Symptomatic treatment:
 - ☛ Fatigue: give flouxetine.
 - ☛ Spasticity: give muscle relaxant.
 - ☛ Bladder: give anticholinestrase.
 - ☛ Ataxia or tremors: give Buspirone

Alsayed Dawoud

Treatment of multiple sclerosis:

(MRI & Gadolinium)

Treatment of acute exacerbations:

- ① Pulse steroid therapy by **methyl prednisolone** 1 gm/D for 3 to 5 days (I.V) then continue (prednisone) $\rightarrow$ 1 mg/kg/day orally (week)
- ② plasmapheresis: if not respond to corticosteroids.

Prevention of relapse:

- Interferone.
- Azathioprine.

$\rightarrow$ IgG عشان
IgM كمين على البجاء $\rightarrow$ IgM

Symptomatic treatment:

- Fatigue: give flouxetine.
- Spasticity: give muscle relaxant.
- Bladder: give anticholinestrase.
- Ataxia or tremors: give Buspirone

MS

$\rightarrow$ Restore the normal Immunity

Immuno-Modulatory:

- $\downarrow$ Frequency and Severity of relapses.
- delay progression to disability.
- interferon beta 1-a } DMAMS.
- Glatiramer acetate 1-b } Disease-Modifying agents for MS.
- Mito Xantrone.
- Alemtuzumab.

ImmunoSuppressive

MAC

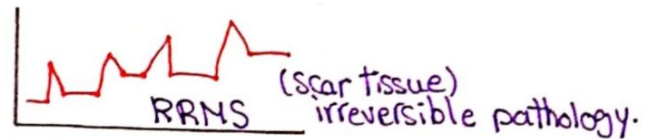
- Methotrexate
- Azathioprine
- Cyclophosphamide.

Clinical patterns: v. I.

1. Relapsing Remitting (RRMS) 85%

75% $\rightarrow$ attack & leave permanent deficits, followed by remission. OR

10% $\rightarrow$ attacks which don't leave permanent deficits followed by remission (Benign MS)



Benign MS

Atsayed Dawoud
Normal myelin sheath after attack
(No scar tissue)

2. Progressive:

- PPMS: From onset $\rightarrow$ continuous deterioration $\rightarrow$ no Remission
- SPMS: 1st RRMS $\rightarrow$ continuous deterioration, "

PPMS

SPMS

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3. progressive relapsing (PRMS)

Steady deterioration & onset + superimposed attack



Epilepsy

Definition:

It is a paroxysmal disorder characterized by recurrent transient disturbances of the brain functions (motor, sensory, psychic or autonomic) associated with EEG changes with or without disturbed consciousness.

Æ:

1) Idiopathic :

☞ +ve FH., start in 1st decade

- a) General mal epilepsy
- b) Petit mal epilepsy
- c) Myoclonic epilepsy

{ the commonest }

2) Symptomatic:

☞ *Epilepsy is only a symptom of underlying brain lesion.*

I. Local:

- ✎ **Congenital** : cerebral palsy
- ✎ **Traumatic** : birth trauma, head injuries
- ✎ **Inflammatory**: meningitis, encephalitis, brain abscess, neurosyphilis
- ✎ **Vascular** : hge, thrombosis, embolism
- ✎ **↑ICT** : tumor, brain abscess
- ✎ **Degenerative** : pre-senile dementia

II. General causes → 2ry effect on brain

- ☺ **Toxic**: alcohol, botulism, CO₂, cyanide, tetanus
- ☺ **Hypoxic**: pulmonary disease → ↑↑RR → hyper ventilation
→ CO₂ wash → alkalosis → ↓ threshold of brain cells
- ☺ **Ischemic**: Adam's attacks, carotid sinus syndrome, F4, anemia.
- ☺ **Metabolic**: hypo & hyperglycemia, hyponatremia, hypercalcaemia, RF, LCF, HF, Resp.F.

- ☺ **Endocrinal** : DM ,thyrotoxic crisis hypoparathyroidism
- ☺ **Hyperthermia** : sun stroke water intoxication high fever especially in children
- ☺ **Iatrogenic** amphetamine aminophylline ambilhar improper use of antiepileptics
- ☺ **Nutritional** pellagra Beri Beri GABA ↓↓
- ☺ **Hysterical**

Classifications:

- I. **Partial seizures** : symptomatic 2ry
Start focally in one region of the brain
 - a. Simple partial seizures with maintained consciousness
 - b. Complex partial seizures with impaired consciousness
 - c. Partial progressing to generalized tonic clonic convulsions
- II. **Generalized seizures** idiopathic 1ry
Start in both cerebral hemispheres at the same time
 - a. Petit mal typical & atypical
 - b. Tonic clonic tonic clonic
 - c. Myoclonic

Clinical picture:

1. Generalized:

- a. General mal epilepsy old age

1. Pre-ictal :

Phase: aura

Good → warning signs for coming attack

Bad → 3-2 min before the attack

May be somatic, psychic and autonomic.

Gives an idea about the site of the attack

2. Ictal :

3phases seizures

- i. Sudden loss of consciousness about 10 min
- ii. Tonic phase 10sec with the onset of coma
 - Rolling eyeball upward
 - Clenching of teeth
 - Extension of UL & LL
 - Turning of head to one side
 - Res. ms. Contraction → cyanosis
 - Laryngeal ms contraction → epileptic cry
- iii. Clonic phase 2-3min
 - Repetition of contraction & relaxation
 - Micturition
 - Tongue biting

3. Post ictal:

Phase : <24h due to neuronal exhaustion

- ~ Fatigue
- ~ Headache
- ~ Sleepiness
- ~ Weakness
- ~ **Todd's paralysis** → transient paralysis following fit

→ Drug of choice carbamazepine _ tegretol _ phenytoin

b. **Petit mal epilepsy** → in children & improve at puberty

Interruption of stream of consciousness {absence seizures} → no aura

May be precipitated by hyperventilation or photic stimulation.

I. Typical :

Sudden loss of consciousness of short duration “few sec” è cessation of motor activity or speech è blank expressions of the face.

II. Atypical:

- 1) Pyknopsy: high frequency 30-100/day for few sec
Staring look , not fall to the ground
- 2) Akinetic “atonic” : attacks of severe hypotonia
Fall to the ground without warning “no aura”
- 3) Myoclonic: sudden jerky movement of the hand & UL

c. **Myoclonic epilepsy :**

1. Simple myoclonus
 2. È general mal epilepsy
 3. È petit mal epilepsy
 4. Juvenile myoclonic epilepsy :
 - 12-16 Ys –frequent jerks upon awaking & disappear later in morning
 - May be accompanied è generalized tonic-clonic
 - ttt è valpromate for life
- ➡ May occur in ms of face ,palate, extremities
- ➡ There is sudden shock –like involuntary movement

d. **Atonic “astatic” seizures**

- ★ Start in children
- ★ Attacks èout warning &for seconds
- ★ Sudden loss of postural tone &fall to the ground no loss of consciousness
- ★ Response to ttt is poor

2. Partial seizuresA. **Simple:**

No loss of consciousness

according to the site of the excitatory focus in the brain

1. Sensory:

- **General:** {area 1,2,3 irritation } in the form of parasthesia involving one limb (focal) or 1\2 of the body sensory jacksonian fits
 - **Special**
 - Visual hallucinations “formed –unformed “
 - Olfactory &gustatory hallucination “irritation of uncus “
 - Auditory hallucinations
 - Depersonalization –derealisation déjà-vu or Jamai-vu
- phenomena irritation of temporal lobe

2. Motor focal :

Movement of part of a limb or the whole limb

Motor Jacksonian fits: movement involving one side of the body spread in a march course “above down or down above.

B. **Complex**

There is disturbed consciousness

The lesion in the temporal lobe.

1. **Aura**: olfactory & gustatory hallucination emotion & fear of elation
Depersonalization –derealisation déjà-vu or jamais-vu phenomena
2. **Absence** the patient appears distant è starring eye & may not respond to questions
3. **Automatism** here may be a champing of teeth ,licking of lips or semi purposeful movements of limbs the patient may walk & leave the room may become violent
4. **Amnesia** for the attack

- C. **Partial seizures where** simple or complex may progress to generalized tonic-clonic seizures

3. **2dry** : (symptomatic)

1. **Sensory → area 1,2,3**

- Affect part of area → pain & parasthesia part of the body e.g. hand
- Affect all of area → pain & parasthesia of ½ of the body {sensory jacksonian fit}
- Also visual, auditory or olfactory hallucinations may occur.

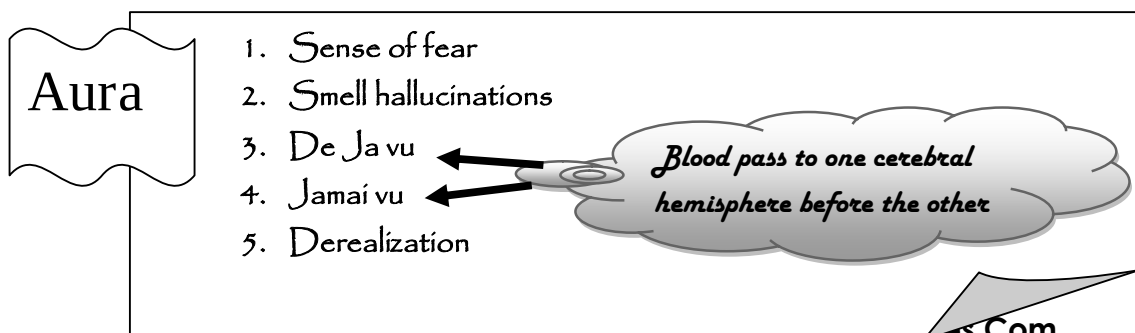
2. **Motor → area 4**

- Affect part of the area → convulsions in part of body {motor jacksonian fit }
- Affect all of the area → convulsions in ½ of body {motor jacksonian fit }

N.B partial simple epilepsy = sensory + motor + no loss of consciousness

Psychic → temporal lobe

- Psychosensory aura only
- Psychomotor 4A ☺
- **Aura**
- **Absence**
- **Automatism**
- **Amnesia**



Investigations:**Aim:**

- ✍ to prove the condition
- ✍ to differentiate symptomatic from idiopathic causes
- 1. **EEG**
- 2. **X-ray, CT, MRI**
- 3. **CSF examination**
- 4. **Lab** : renal functions ,blood sugar , CBC,

Treatment:

I. General :

1. **Modification of life :**

No swimming,

No driving,

Adjust the work of the patient to keep him safe away of accidents

2. **Avoid ppt factors :**

- photic stimulation
- missed drugs (bad ttt)
- high sounds

3. **diet :**

- **No** alcohol
- ketogenic diet : fat induce acidosis

4. **ttt of the cause**

II. specific ttt :

⇒ *Basic principles* ⇐

1. Monotherapy :

Is the best approach to newly diagnosed epileptics ,the drug of choice is selected according tp seizure type

2. Multiple drugs are more expensive & ↑↑ the risk of side effects & drug interaction

3. Proper dose

4. Antiepileptics are discontinued gradually only when the patient has been free from fits for at least 2ys

Side effects:

Allergy, GIT irritation, B.M. depression, status epilepticus, ataxia.

Blood dyscrasias → frequent Bl. Picture should be done.

❖ Depakin → hepatotoxic

❖ Epanutin → gum hyperplasia & hirsutism.

Antiepileptic drugs:

Drug group	Dose	Best indicated
1. Barbiturates: Luminal(phenobarbitone)	100-600mg\day	Broad spectrum anticonvulsant Not for petit mal
2. Hydantoin: epanutin	200-600mg\d	Simple partial motor seiz. Grand mal seiz.
3. Carbamazepine: Tegritol	400-800mg\d	Simple partial motor seiz. Complex partial motor seiz. Grand mal seiz. Not for petit mal
4. Clonazepam: Rivotril	2-6mg\d	Myoclonic seiz. Grand mal seiz.
5. Valproate: depakin	600-1500mg\d	Simple partial motor seiz. Complex partial motor seiz. Petit mal seiz. Grand mal seiz. Myoclonic seiz.
6. Succinimide: Zarontin	500-1000mg\d	Petit mal seiz.
7. New anti-epileptics : – Gabapentin(neurontin) – Vigabatrin(sabril) – Topiramate(topamax) – Lamotrigine(lamictal) – Tiagabine(gabitril)	1200-2400mg\d 2000-3000mg\d 400-800mg\d 200-600mg\d 30-60mg\d	Refractory partial & generalized seiz Refractory partial & generalized seiz Refractory partial & generalized seiz Refractory partial & generalized seiz Refractory complex partial seiz

Status epilepticus

Definition:

Recurrent attacks of generalized convulsions without regain of consciousness.

It is fatal condition as it cause exhaustion, fatigue → any failure

Æ:

Sudden withdrawal of antiepileptic drugs.

C/P:

Generalized convulsions esp. Grand mal epilepsy.

ttt:

1. General:

- a. Managed on **IUC** → intubated or placed on mec. Ventilator.
- b. Guard against → fall from the bed
→ tongue clenching {mouth gage }
- c. general :
O₂ inhalation
monitoring of: vital signs

P_{O2} , P_{CO2} , PH & Bicarb → {ABG }.

CBC, antiepileptics level, Glc, electrolytes → venous sample

Record ECG daily .

Start infusion of 500 ml normal saline

- d. cerebral dehydrating agents {mannitol –lasix}

2. medical :

- 1) Epanutin “phenytoin”: 15mg/kg infusion by a rate of 50ml/min
C.I: cardiac arrhythmia & recent MI
- 2) Diazepam 5mg I.V. to be repeated every 5 min max 20mg
- 3) If no improvement → phenobarbiton 2200mg infusion very slowly
- 4) In resistant cases general anesthesia may be used.

Coma

Introduction:

COSCIOUSNESS is a state of awareness of self & surrounding.

The state of consciousness depends on :

1. intact ARAS (present in brain stem , hypothalamus , thalamus)
2. intact cerebral cortex

So, small lesion of ARAS OR extensive diffuse lesion of CC → Coma

Etiology:

A: Intracranial causes (with signs of lateralization)

1. Trauma : cerebral contusion
2. tumor : meningioma
3. vascular : thrombosis embolism hge ,HTN encephalopathy
4. Inflammatory : meningitis, encephalitis brain abscess
5. epilepsy

N.B

All intracranial causes → signs of lateralization except HTN encephalopathy

B: Extracranial causes:



1. **Toxic**: atropine ,aspirin ,alcohol
 - ↳ Barbiturates
 - ↳ CO poisoning
 - ↳ Morphine
2. **Hypoxic**: Pulmonary disease.
3. **Ischemic** : cardiac arrest ,MI ,hypotension
4. **Metabolic** : DM ,LCF ,RF ,collagen disease
5. **endocrine** : DM ,Hyper&Hypothyroidism ,Hyper&Hypoparathyroidism ,Addison's crisis
6. **Fever** (febrile coma)

Febrile coma:

- **Infective** : encephalitis , meningitis ,UTI ,pneumonia
- **Vascular** : subarachnoid hge
- **Toxic**: Atropine ,aspirin ,Halothane(malignant hyperthermia).
- **Metabolic** : Hyperthyroidism ,Addisonian crisis ,DKA.
- **Sun stroke**

Diagnosis of a case of coma:

1. **clinical exam.:** **1** Degree
 2 Etiology

2. Investigations for the cause.**Degree of coma**

I: Old classification:

- 1. Drowsiness**(lethargy) :state of disturbed consciousness in which the patient can be aroused to call
- 2. Stupor:** **patient** can be aroused by only vigorous &repetitive painful stimulation & return to deep sleep when not continually stimulated.
- 3. Semi coma** : complete loss of consciousness with a response only at the reflex level
- 4. coma** : un arousable , unresponsiveness to all stimuli

II: Glasgow coma scale :

Eye opening	Verbal response	Motor response
Spontaneous...4	Oriented5	Obey commands...6
To speech3	Confused4	Localize to pain5
To pain2	Inappropriate words.....3	withdraw from pain 4
None.....1	Inappropriate sounds...2	Flex to pain3
	None1	Extend to pain2
		None1

Mild : 13-15

Moderate: 9-12

Sever : ≤ 8

Approaches to Diagnosis:

I. **History** : from relatives

- ★ About mode of onset :
Sudden: Hge.& Embolism

Gradual: Thrombosis & DKA
- ★ History of DM _ Epilepsy _ Drug intake

II. **Examination** :

☑ **Temp:**

- Hyperthermia :see febrile coma
- Hypothermia : hypothyroidism _ Morphine

☑ **Pulse:**

- ↑↑ hyperthyroidism
- ↓↓ hypothyroidism

☑ **BL.Pr:**

- ↑↑ in HTN encephalopathy
- ↓↓ in Addisonian crisis

☑ **Odour of breath**

- Acetone→ DKA
- Uriniferous→CRF

☑ **Respiration:**

- Rapid deep respiration → DKA
- Slow deep respiration.→ Morphine

☑ **Skin:**

- Dry→ DKA
- Moist→hypoglycemic coma
- Needle markers→ addiction

☑ **Pupil:**

- Dilated : atropine poisoning (bilateral)
- Constricted : morphine , metabolic coma

☑ **CNS: Signs of lateralization ☠☠**

- *Unequal pupils*
- *Facial asymmetry*
- *Turning of the head to one side*
- *Unilateral hypo. Or hypertonia*
- *Unilateral Babinski*
- *Unilateral jacksonian fits*
- *Asymmetrical deep reflexes.*

Investigations:

Intra Cranial: CT, MRI, CSF.....

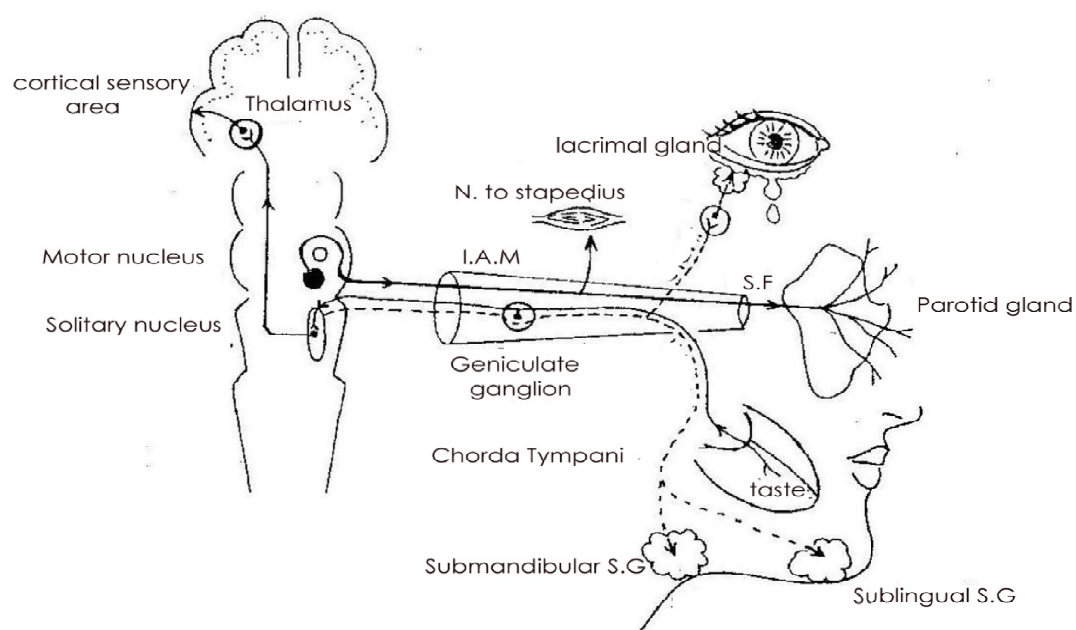
Extra Cranial : *in basis of the most probably cause*

Treatment:

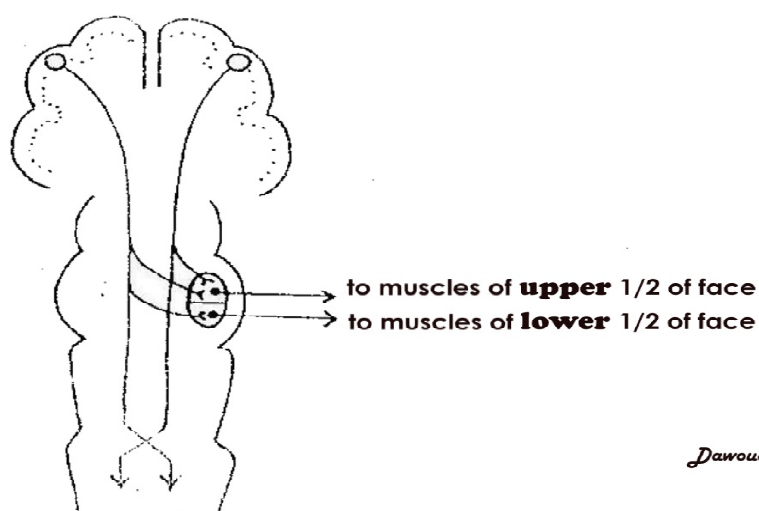
*General care: 5B →see hemiplegia
ttt of the cause.*

Facial nerve

Anatomy of the Facial Nerve



Pyramidal supply to facial nucleus



Dawoud 2009

In Capsule Series

In Capsule Series

Neurology

It is a mixed nerve as it contains:

- 1) Motor fibers to the following muscles:
 - ⇒ Orbicularis Oculi
 - ⇒ Orbicularis Oris
 - ⇒ Frontalis muscle
 - ⇒ Buccinator muscle
- 2) Sensory fibers:
 - ⇒ Taste sensation from anterior 2/3 of the tongue
- 3) Parasympathetic fibers:
 - ⇒ for Salivary and lacrimal glands

Clinical picture:

UMNL (affecting the pyramidal tract)

1. Obliterated nasolabial folds
2. Dropping of angle of the mouth
3. Accumulation of the food behind the cheek
4. Inability to blow the cheek
5. Inability to show the teeth ☹

LMNL (affecting the motor nerve or the facial itself)

In addition to the above;

1. Inability to close the eye → when he tries to close his eye → eye ball rolls upward
(*Bell's phenomenon*)
2. Inability to rise his eyebrows

UMNL	LMNL
Features :	
◆ Hemiplegia on the opposite side ◆ Paralysis of the muscle of the lower ½ of the face on the opposite side. ◆ Intact emotional movements (Supplied by extra pyramidal fibers.)	◆ Crossed hemiplegia ◆ Paralysis of muscles of upper and lower ½ of the face on the same side (see brain stem hemiplegia) ◆ Paralysis of emotional movements (pyramidal and extra pyramidal)

 **facts see page 1**

N.B: Facial nucleus;

Upper $\frac{1}{2}$ bilaterally supplied by pyramidal tract of both sides.

Lower $\frac{1}{2}$ unilaterally supplied by pyramidal tract of the opposite side.

Site of lesion in LMNL

Nuclear lesion: (in pons)

⇒ Paralysis of all facial muscles and intact taste and salivation.

Cerebello-pontine Angle lesion.

⇒ Paralysis of facial muscle and diminished taste on anterior 2/3 of the tongue, salivation & lacrimation.

Facial canal lesion:

⇒ Paralysis of all facial muscles

⇒ And according to the site of the lesion;

✗ If **chorda tympani** is involved →(↓↓ taste sensations from 2/3 of tongue & salivation)

✗ If **GSPN** is involved →(↓↓ lacrimation)

BELL's palsy ☠☠

Definition:

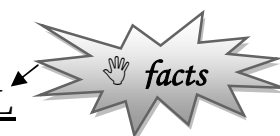
Non-suppurative inflammation of the facial nerve just before the terminal end of facial canal →acute paralysis of the face

Etiology: Idiopathic

1. Vascular: Air drafts →V.C →Edema →Compression on facial nerve
2. Viral: Herpes Zoster.
3. Autoimmune

Clinical picture:

- # Facial nerve paralysis with criteria of **LMNL**
- # Pain usually behind the ear
- # There may be, impaired sensation of taste on ant.2/3 of the tongue on the same side



Treatment:

Medical: (recovery rate 80%)

- * Cortisone (prednisolone tab. 30 mg/day)
- * Vasodilators and vitamin B complex
- * Protection of the cornea (eye ointment)
- * Physiotherapy

Surgical: (for the remaining 20%)

- * Decompression of facial nerve in the facial canal
- * Grafting

HOW to answer

Q: Facial Nerve Paralysis??

1. Anatomy
2. Differences between UMNL & LMNL
3. Clinical picture
4. Site of lesion in LMNL
5. BELL's palsy

*Studying medicine without teacher is like sailing without a boat, and studying
Internal medicine without **Dr/Ahmed M.Mwafy** is like not going to the sea at all.
Alsayed dawoud
Kasr-Alainy school of medicine.*

LMNL Lesions of facial N.

Site of Lesion :

Causes :

Pontine lesions (Nuclear)

- 1- paralysis of facial muscles
- 2- No impairment of taste Sensation
- 3- No impairment of salivation or lacrimation
- 4- May be other LMN cranial palsies on the same side or hemiplegia on the opposite side

- 1-vascular
 - vertebral-basilar insufficiency
 - millard-Gubler §
- 2- infective
 - Encephalitis, poliomyelitis
- 3- Neoplastic:
 - Glioma
- 4- Demyelinating: DS.

Cerebello-pontine Angle lesion

- 1- paralysis of facial Ns.
- 2- ↓ Taste on ant. 2/3 of Tongue
- 3- ↓ Salivary and Lacrimal secretions.
- 4- Associated Cr. 5, 6, 8 palsies on same side
- 5- Ipsilateral cerebellar ataxia

- 1- infective
 - Basal meningitis.
- 2- Neoplastic
 - Acoustic Neuroma, meningioma.

Facial canal lesion

- 1- paralysis of facial Ns.
- 2- ↓ Taste on ant. 2/3s of Tongue, and ↓ salivation • if chorda Tympani is involved.
- 3- Diminished lacrimation if the greater superficial petrosal nerve is involved.
- 4- Hyperacusis if nerve to stapedius is involved.

- Traumatic → Fracture base
- Infective → otitis media, HZ
 - Neoplastic → Facial Neuroma
 - Bell's palsy.

Extra-cranial lesions

(after its exit from stylomastoid foramen)
paralysis of facial muscles only.

- 1- Neuropathy: DM, leprosy
- 2- Myopathy: fascioscapulohumeral
- 3- Myasthenia.
- 4- invasion by Tumor (parotid).
5. injury to the nerve during surgery.

Complications of Bell's palsy ?

1- Crocodile tears. Flow from affected eye during eating.

- Excessive tear

• Explanation : regenerating fibers to the submandibular gland become misdirected and reach the **Neurology** lacrimal G.

2- Corneal ulcers.

3- Incomplete Recovery : This may results in residual facial asymmetry.

Vertigo

Def:

- It is the sense of rotation of the body in steady surrounding or the reverse i.e.:
- The body may be felt to rotate or fall while the surrounding are steady OR
- The surrounding themselves appear to rotate around the body.
- It is aggravated by movements of the head and it persists in all positions: Sitting, standing or supine.
- associated with autonomic manifestations in the form of: Nausea, vomiting, pallor, Sweating.
- Differentiated from dizziness, where the unsteadiness is not associated with a sense of rotation.

Etiology:

1. Labyrinthine

- a) physiological: Sea sickness, Car sickness
- b) pathological: labyrinthitis, Meniere's disease

2. Peripheral Nerve:

- Cerebello-pontine Angle lesion as acoustic Neuroma
- vestibular Neuritis.

3. Brainstem

- VB Artery insufficiency
- PICA occlusion
- DS • encephalitis

4. Cerebral: ↑ ICT.

5. psychogenic

- occur in acute anxiety.
- Responds to Tranquillisers.

6. Benign positional vertigo:

- Usually Idiopathic OR due to inner ear disease
- associated with Nystagmus and is usually by changing the position such as turning in bed.
- usually mild, transient, lasting less than 30 seconds.

Treatment:

- Treatment of the Cause.
- Cinnarizine (Stugeron) and Dramamine may be used.

Vestibulo cochlear Nerve

2 Divisions:

	cochlear	vestibular
Function	Hearing	Equilibrium.
Lesion	- Irritative: Tinnitus - Destructive: Deafness.	- Vertigo - Nystagmus - Incoordination in the same side of the body.

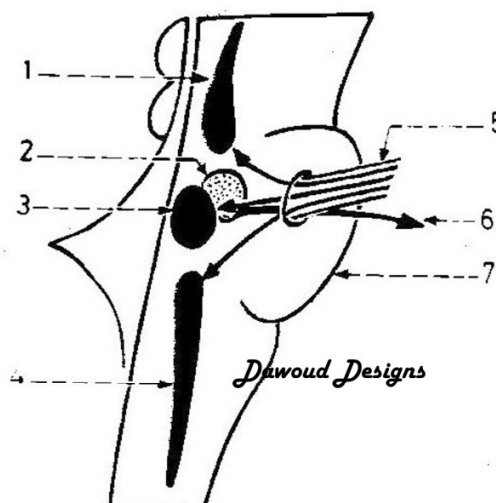
Anatomy of the Trigeminal Nerve

NUCLEI OF TRIGEMINAL NERVE

These are one motor and 3 sensory nuclei (mesencephalic, main sensory and spinal).

1. mesencephalic nucleus (in the midbrain).
2. motor nucleus (in the pons).
3. main sensory nucleus (in the pons).
4. spinal nucleus (in the medulla oblongata).
5. sensory root of trigeminal nerve (its fibres end in the 3 sensory nuclei).
6. motor root (its fibres arise from the motor nucleus).
7. pons.

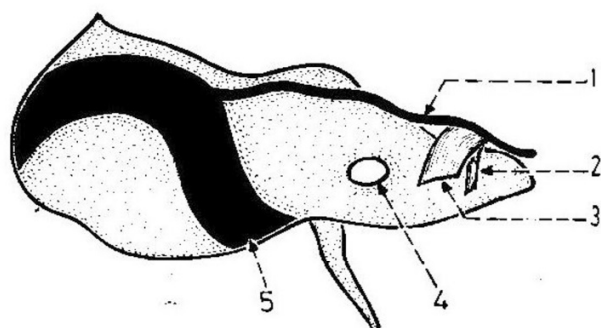
* The mesencephalic nucleus is concerned with proprioception, the main sensory nucleus with touch and pressure while the spinal nucleus is concerned with pain and temperature.



ROOTS OF TRIGEMINAL NERVE

The nerve has a large sensory root and a smaller motor root. The 2 roots cross over the upper border of the petrous bone near its apex deep to the superior petrosal sinus.

1. superior petrosal sinus.
2. motor root.
3. sensory root.
4. internal acoustic meatus.
5. sigmoid sinus.



Lesions of the Trigeminal Nerve

Sensory affection:

1) Peripheral lesions;

- ☞ Loss of sensations on the same side of the face according to the affected branch.

2) Central lesions;

Pontine lesion:

- ☞ Weakness of the muscles of mastication
- ☞ Loss of touch sensation over the face with preservation of pain & temp. sensations.

Lesions in the medulla:

- ☞ Loss of pain and temp. with preservation of touch e.g:
Syringobulbia & PICA & Tabes dorsalis

N.B.

the upper part of the spinal nucleus supplies the lower face (Mandibular branch)
And the outer part supplies the inner face & Vice Versa

So, in Syringobulbia: dissociated sensory loss starting from the *outer part* of the face.

Tabes dorsalis: starting from the *central area* of the face (around the nose)

Motor affections:

- ☞ Weak muscles of mastication of the same side
- ☞ **Masseter** and **Temporalis** are palpated during clenching the teeth and their power can be estimated.
- ☞ **Pterygoids**: ask the patient to open his mouth while you fix his head.
 - ☞ Unilateral paralysis: deviation of the jaw to the diseased side
 - ☞ Bilateral paralysis: no deviation but, inability to open the mouth against resistance.

Reflex affections:

- ☞ Loss of the corneal reflex → (afferent 5 & efferent 7)
- ☞ Loss of palatal reflex → (afferent 5 & efferent 10)
- ☞ Exaggerated jaw reflex → (afferent 5 & efferent 5) in bilateral UMNL

N.B.

Jaw reflex: when the jaw is tapped while the mouth is slightly opened nothing will appear as the reflex is too weak to be detected.

☑ Pyramidal tract lesion above the trigeminal nucleus causes the reflex to be **exaggerated**.

Trigeminal Neuralgia (Tic douloureux)

Definition & C/P:

- ✱ Electric-shock like, lancinating pain with sudden onset, sudden termination along the distribution of one or more of the divisions of the trigeminal nerve usually 2nd & 3rd divisions
- ✱ More common in D.S, DM. and Alcoholism
- ✱ More common in ♀ ☺
- ✱ Pain is commonly evoked by shaving, smoking, talking, brushing the teeth & may also occur spontaneously

Investigations: Nothing ☞

Treatment:

Medical:

1. Tegretol 200-400 mg t.d.s
2. Phenytoin
3. Clonazepam
4. Morphine 10 mg IM. May be used

Surgical:

1. Alcohol injection into the nerve
2. Microvascular decompression

☠☠ D.D of Facial pain:

1. **Trigeminal neuralgia.**
2. **Diseases of teeth, sinuses, ear, nose or the throat.**
3. **Migrainous neuralgia(Cluster headache)**
4. **Post herpetic neuralgia**
5. **Temporo-mandibular arthritis**
6. **Cervical spondylosis**
7. **Temporal arteritis**
8. **Myocardial ischemia**

Trigeminal Neuralgia (Tic douloureux)

P:

Shock like, lancinating pain with sudden onset, sudden termination
distribution of one or more of the divisions of the trigeminal nerve
1st & 3rd divisions

Common in D.S., DM, and Alcoholism

Common in ♀

Commonly evoked by shaving, smoking, talking, brushing the teeth

Also occur spontaneously

Nothing

★ by History only.

Unknown etiology

1 May be → nerve root

early: irritant

Late: destructive lesion.

①

Compression by

• Tumor ✓

• Aneurysm ✓

• Vascular anomaly

abnormal vascular course of

SCA

C/P → Pain

200-400 mg t.d.s

Carbamazepin

500-1000 mg/day oral

10 mg IM. May be used

injection of affected sensory root.

injection into the nerve

vascular decompression

Facial pain:

Trigeminal neuralgia.

Pain of teeth, sinuses, ear, nose or the throat.

Cluster neuralgia (Cluster headache)

Herpetic neuralgia (virus in T. ganglion)

Temporomandibular arthritis, Costen's Syndrome

Spinal spondylosis

Temporal arteritis

Site + reference: usually 2nd, 3rd branches
unilateral.

character + duration:

• Severe agonizing, stabbing, burning.

• Several Seconds to several minutes

• Recurrent: Free in b/n attacks.

precipitated by movements of jaw.

Relieved by: Spontaneously or potent analgesic

Association: muscle spasm of facial muscles.

→ pain worse in morning +
(Sinusitis) Tenderness over sinus

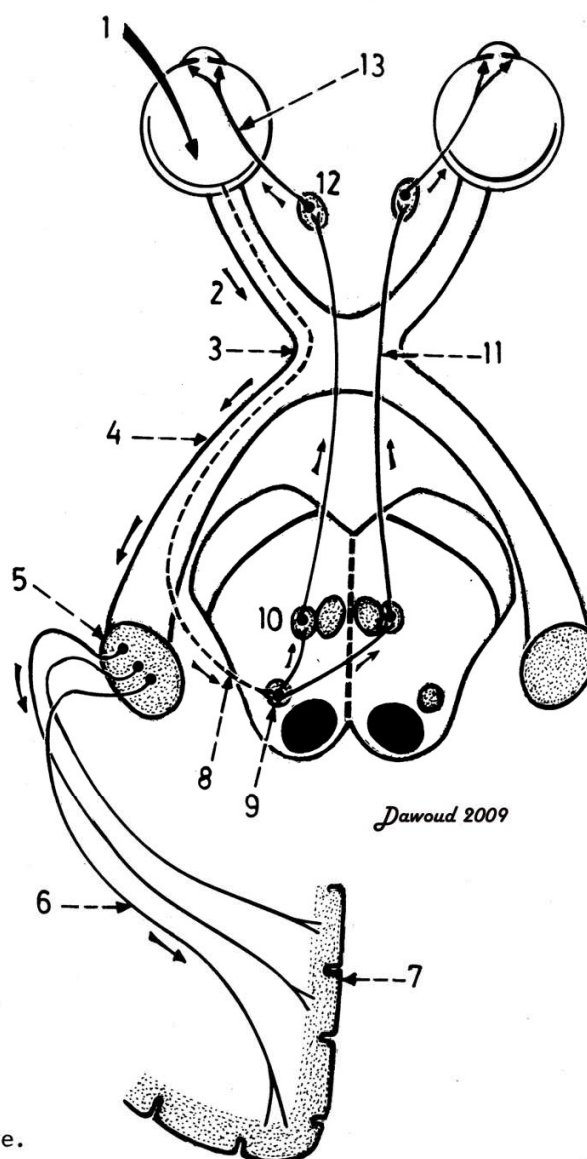
In Capsule Series

Pathway of Vision and light reflex

The pathway of vision is as follows: retina, optic nerve, optic chiasma, optic tract, lateral geniculate body, optic radiation and occipital cortex. The pathway of light reflex is as follows: retina, optic nerve, optic tract, pretectal nucleus, Edinger-Westphal nuclei of both sides, oculomotor nerves of both sides, ciliary ganglia of both sides and postganglionic fibres to the sphincter pupillae muscle of the iris of both eyes.

1. light falling on the retina.
2. optic nerve.
3. optic chiasma.
4. optic tract.
5. lateral geniculate body.
6. optic radiation.
7. occipital cortex.
8. fibres of light reflex leaving the optic tract.
9. pretectal nucleus.
10. Edinger-Westphal nucleus.
11. oculomotor nerve (preganglionic fibres).
12. ciliary ganglion.
13. postganglionic fibres to the sphincter pupillae muscle of iris.

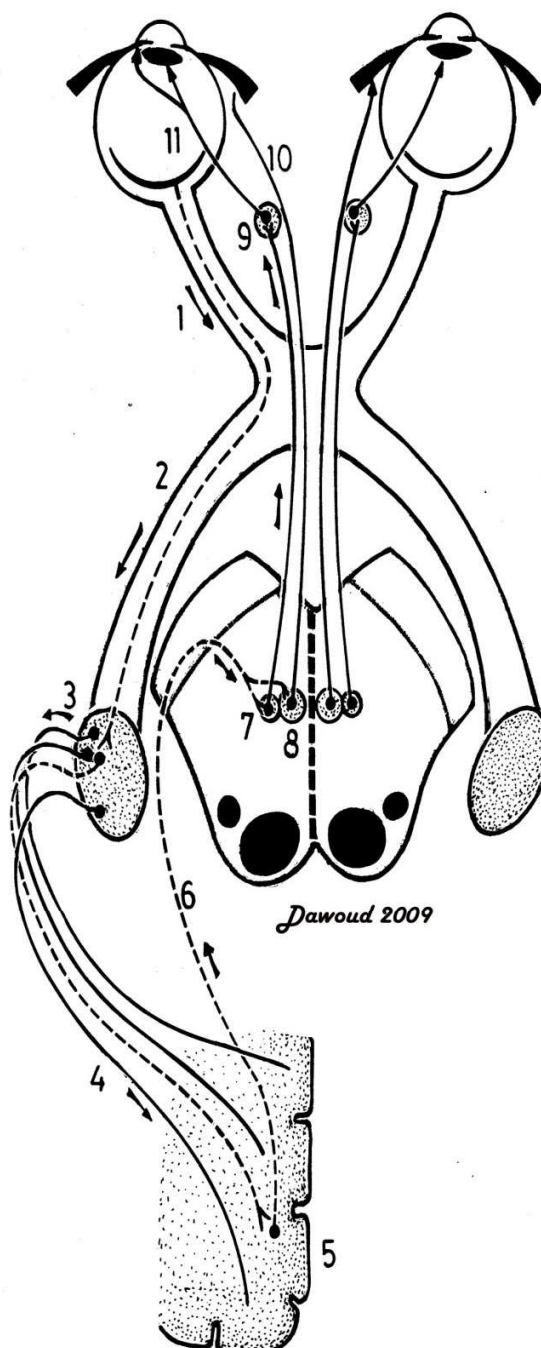
* In light reflex, exposure of one eye to bright light results in contraction of the pupil of the same as well as of the opposite eye.



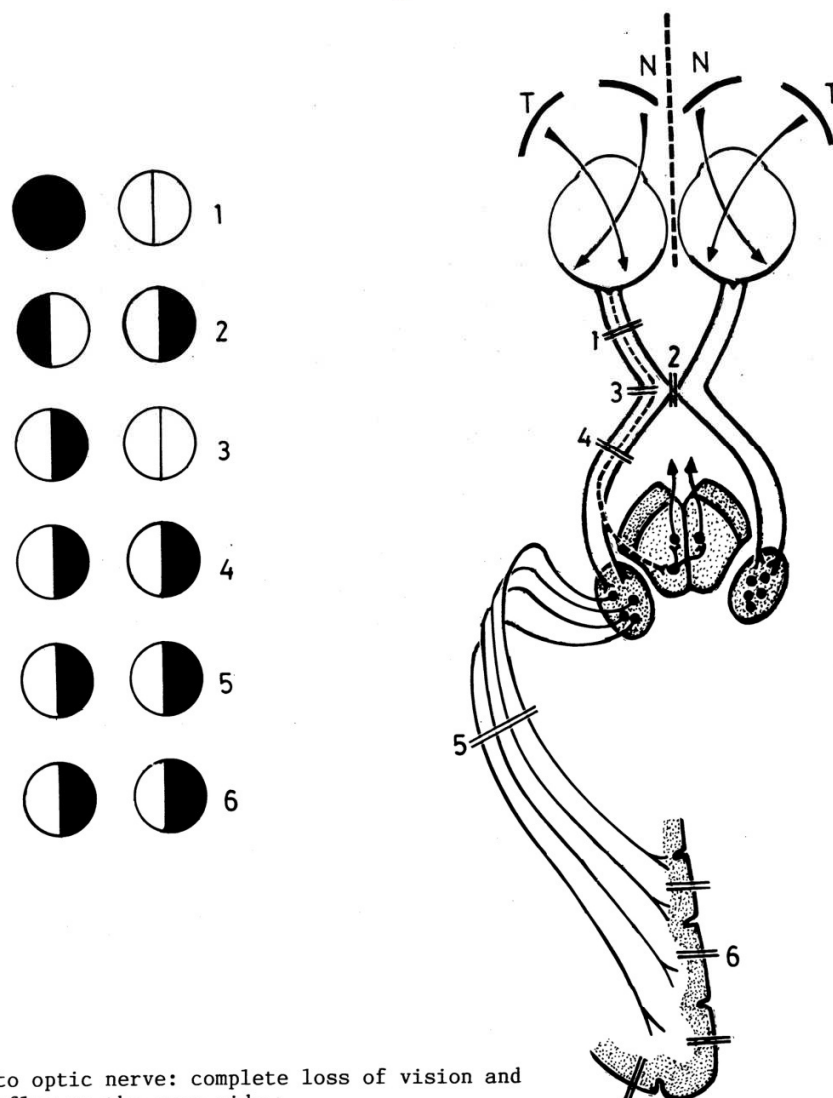
Accomodation Reflex

In accomodation reflex the eyes converge, the ciliary muscle contracts to modify the shape of the lens and the constrictor pupillae muscle of the iris contracts to constrict the pupil. Its pathway is as follows: retina, optic nerve, optic tract, lateral geniculate body, optic radiation and visual area in the occipital cortex. Efferent fibres from the occipital cortex pass to the oculomotor nucleus and then through the oculomotor nerve to the extra- and intra-ocular muscles.

1. optic nerve.
2. optic tract.
3. lateral geniculate body.
4. optic radiation.
5. occipital cortex.
6. efferent fibres to the oculomotor nucleus.
7. Edinger-Westphal nucleus.
8. oculomotor nucleus.
9. ciliary ganglion.
10. fibres of oculomotor nerve to extra-ocular muscles.
11. postganglionic fibres to intra-ocular muscles.



lesions of Visual pathway



1. lesion to optic nerve: complete loss of vision and light reflex on the same side.
2. lesion to the middle of optic chiasma: bitemporal hemianopia.
3. lesion to the lateral part of optic chiasma: ipsilateral nasal hemianopia and loss of light reflex on the same side.
4. lesion to optic tract: contralateral homonymous hemianopia and loss of light reflex.
5. lesion to optic radiation: contralateral homonymous hemianopia without loss of light reflex.
6. lesion to visual area of occipital cortex: contralateral homonymous hemianopia without loss of light reflex.

* Bitemporal hemianopia = loss of temporal fields of vision on both sides.

* contralateral homonymous hemianopia = loss of temporal field of opposite side and nasal field of same side.

Sciatica

Definition:

It is a pain along the distribution of sciatic nerve → back of thigh, leg and foot.

Causes:

- ① Acute disc prolapse (traumatic)
- ② Lumbar Spondylosis
- ③ Malignant pelvic tumor
- ④ Sciatic nerve neuritis as in D.M.

N.B

the commonest cause of sciatica is disc prolapse MCQ

Clinical picture:

Symptoms:

Pain along the course of sciatic nerve ↑↑ by cough, straining, stretching

Signs:

1) Sensory:

⇒ hypoesthesia along sciatic nerve

⇒ +ve signs of meningeal irritation as this lead to traction on roots of sciatic nerve,
pt. can't elevate the leg up to 90° without pain

2) Motor: LMNL in m_s supply by the nerve

3) Back pain

Investigations:

- ✎ X-ray
- ✎ Myelography
- ✎ CT Scan

Treatment:

1. Medical ♠ NSAID

♠ M_s Relaxant: Norflex® & Glifarelix®

2. Physiotherapy

3. Surgery: Decompression laminectomy

Meningitis

Definition:

- ⇒ It is an inflammation of the membrane covering the CNS
- ⇒ Inflammation of the dura is rare & is known as pachmeningitis hypertrophica \$.
- ⇒ Inflammation of the pia & arachnoid is more common & known as leptomeningitis

Classification:

① **Acute pyogenic meningitis** *the CSF contains P.N.L*

- ✎ Meningiococci
- ✎ Haemophilis
- ✎ Streptococci
- ✎ Pneumococci

② **Subacute lymphocyte (non-pyogenic)** *the CSF contains lymphocytes*

- ✎ T.B.
- ✎ Viruses e.g. : herpes
- ✎ Fungi

Mode of infection:

- ✎ Blood stream from infection of other sites
- ✎ Direct from:
 - * Trauma: L.P. or penetrating wound
 - * Septic focus: sinusitis, otitis media, encephalitis

Meningococcal meningitis (acute cerebrospinal fever)

Clinical picture:

Symptoms:

- Acute onset of fever, vomiting, blurring of vision.
- Stiffness of the neck. (DD; subarachnoid hge. *Headache* then *Stiffness*).

Signs:

☐ General:

- ☑ Fever 38-39 C or more.
- ☑ Rapid then slow pulse with ↑ ICT.
- ☑ Blood pressure normal except with sever cases.
- ☑ **Kgic skin** rash on the trunk due to capillary fragility (*purpura fulminans*)

Water house friedreichson's \$

- ◆ *purpura due to ↑ cap. fragility → purpura fulminans*
- ◆ *Adrenal gland hge → acute adrenal insufficiency & shock*

☐ Signs of meningeal irritation:

1. **Neck rigidity:** → passive flexion of the neck is difficult i.e.:
elevation of the neck and trunk together.
2. **Neck retraction.**
3. **Brudzinski sign:**
 - ☞ Neck flexion → flexion of both knees
 - ☞ Flexion of one hip → flexion of the other knee
4. **Kernig's sign:**
 - ☞ patient in the supine position with his hip & knee flexed at 90
 - ☞ Try to extend the knee severe pain.
5. **Leg elevation:** → also +ve in sciatica due to affection of the root.
(*Normal patient can elevate his limb up to 90 degree*).

☐ Signs of neurological deficit :

- ☑ Clouding of consciousness.
- ☑ Transient affection of the cranial nerve
- ☑ Convulsions.

Complication of meningitis :

1. **Neurological:** hydrocephalus - deaf - focal lesion as hemiplegia, aphasia with affection of the cortex (meningoencephalitis)
2. **Cardiac:** pericarditis - endocarditis.
3. **Eye:** conjunctivitis - keratitis iridocyclitis.
4. **Genitourinary:** orchitis - nephritis.
5. **Joint:** purulent arthritis.

Investigations:

☠ **CSF examination**

- a. ↑pressure
- b. ↓sugar (*meningococci consume sugar*)
- c. proteins (*Being exudate*)
- d. Purulent
- e. ↑PNL (*being pyogenic infection*)

☠ **Blood** → leucocytosis

Treatment:

- 1) **Crystalline penicillin G:** 24 million units/24 hrs 1/4 dose rapidly
- 2) **Chloramphenicol:** 100 mg/kg can be used in Patient allergic to penicillin
- 3) **3rd genera on cephalosporin:** can be used → Cefotaxime (claforan ®)
- 4) **Mannitol:** for brain edema, **Heparin:** for DIC.

T.B. meningitis

- Onset is insidious & preceded by Signs of toxemia
- Signs of meningeal irritation → mild.
- Usually there is a primary tuberculous focus.
- CSF → ↑ pressure & proteins - ↓sugar - cloudy - cells are mainly lymphocytes and ↓↓ Cl markedly.
- **ttt.** Rifampicin & INH.

Encephalitis

Definition:

It is an inflammation of the brain substance.

When the spinal cord is involved it is called → Encephalomyelitis

Etiology:

Primary → is caused by organisms which primarily attack the CNS:

→ Rabies - Polio - Inclusion body virus.

Secondary → caused by organism which don't primarily attack the CNS but invade outside the brain then invade the nervous system:

→ **Viral:** mumps, herpes (commonest H. Simplex)

→ **Parasite:** malaria.

→ **Bacterial:** typhoid

Clinical picture:

① The onset is usually acute with ***influenza like symptoms*** including mild fever, headache

② **Neurological defect:**

- ⇒ Somnolence
- ⇒ Ocular paralysis
- ⇒ extra pyramidal manifestation
- ⇒ Mental manifestations
- ⇒ Signs of meningeal irritation
- ⇒ Monoplegia or paraplegia

Treatment:

1. Proper nursing.
2. Acyclovir ® therapy for H. Simplex parental.
3. ACTH. 50 IU/d. IM → releases cortisone.
4. Antibiotics
5. Dehydrating measures.

Motor neuron disease

Definition :

- MND is defined as **pure** progressive **degeneration of motor neurons**. It affects the UMN and/or LMN (The anterior horn cells of the spinal cord & motor nuclei of the cranial nerves).
- In MND, no sensory involvement, no bladder manifestation, no ocular affection, no sexual dysfunction. *Only the motor system is affected.*
- Depending on the distribution of involvement, MND is subdivided into 3 main groups :
 1. **Combined UMN & LMN affection :** **Amyotrophic lateral sclerosis (ALS)**
It is the **commonest** type.

2. UMN affection :

- A. Lateral sclerosis : Purely UMN in the spinal cord
- B. Pseudobulbar palsy : UMN in the cortex and brainstem

3. LMN affection :

- A. Progressive muscular atrophy (PMA) : AHCs in the spinal cord
- B. True bulbar palsy : motor nuclei of the cranial nerves.

Etiology :

- Most cases of MND are sporadic (90%). Only 10% are familial.
- The cause of MND is still unknown. It may be due to **glutamate mediated neurotoxicity**.
Glutamate is a neurotransmitter that may play a role in destruction of motor neurons.

Clinical picture :

1. UMN affection :

A. Lateral sclerosis : Purely UMN in the spinal cord, rare

- Bilateral UMN of the limbs (Paraplegia or quadriplegia)
- The diagnosis can be made only after other conditions are excluded.
- It may progress to classic ALS.

B. Pseudobulbar palsy : UMNL in the cortex and brainstem (corticobulbar tract)

Spasticity of the bulbar muscles (jaw, face, soft palate, pharynx, larynx and tongue).

- Bulbar manifestations : e.g. Dysphagia, Dysarthria
- Donald Duck dysarthria : that sounds as if the patient is trying to squeeze out words from tight lips.
- Tongue : **spastic** tongue, no wasting initially, no fasciculations.
- Exaggerated pharyngeal (gag) reflex.
- Exaggerated jaw reflex.
- Emotional lability.
- Examination may reveal UMNL of the limbs.

2. LMN affection : Here, the disease affects the AHCs in the spinal cord or motor cranial nuclei in the brain stem**A. Progressive spinal Muscular Atrophy “PMA” :**

- It affects AHCs in the spinal cord (mostly lower cervical & lumbar)
- PMA is associated with LMN signs (wasting, weakness and fasciculations) which often begin in the small muscles of the hands or feet then spread.

B. Bulbar palsy : LMN lesion of X, XI, XII cranial nerves in the medulla.

- Bulbar manifestations : Dysphagia, Dysarthria, Nasal regurgitation, Nasal speech.
- The tongue is **wasted** and shows **fasciculations**.
- Absent palatal and pharyngeal reflexes.
- There is no quadriplegia or emotional changes.

Pseudo-bulbar palsy	Bulbar palsy
○ Bilateral UMNL of X, XI, XII cranial nerves resulting from bilateral lesion of corticobulbar pathways .	○ LMN lesion of X, XI, XII cranial nerves in the medulla .
○ Spastic tongue, <u>no fasciculations</u> . It can't be protruded, lies on the floor of the mouth.	○ Wasted <u>fasciculating</u> tongue
○ Jaw reflex : exaggerated	○ Jaw reflex normal or absent
○ pharyngeal (gag) reflex : exaggerated	○ Gag reflex : absent
○ Spastic dysarthria.	○ Nasal regurgitation, nasal tone due to escape of air through nose
○ Associated with quadriplegia	○ No quadriplegia
○ Emotional lability may be present	○ No emotional lability.
○ Causes include : - Bilateral cerebral lacunar infarction. - Multiple sclerosis. - Motor neuron disease. - High brainstem tumors.	○ Causes include: - Motor neuron disease. - Poliomyelitis - Syringobulbia. - Guillain Barre syndrome. - Myasthenia.

3. Combined UMN & LMN affection : **"Amyotrophic lateral sclerosis, ALS"**

(Lou Gehrig disease, Lou Gehrig - baseball player. He died in 1941 as a result of ALS)

- It is the commonest type of MND (85% of cases). Most cases are sporadic (90%). Only 10% are familial (autosomal dominant).
- Age : usually > 50 years, More common in men (1.5 : 1).
- Painless **progressive muscle weakness** is the hallmark feature.
- There are **mixed** manifestations of UMN & LMN : *"Tonic atrophy"*
 - **LMN** : Muscle **atrophy** & fasciculations.
 - **UMN** : Hyperreflexia, **hypertonia** (spasticity) & +ve Babinski.
- Usually first noted in the LL or UL then spreads to other muscle groups. Foot and hand deformities are seen as a result of tendon imbalance & joint contractures.
- There is often a **mixed bulbar & pseudobulbar picture** (e.g. wasted fasciculating tongue with a spastic palate).
- No sensory involvement, no bladder manifestation, no ocular affection, no sexual dysfunction. Only the motor system is affected.
- **Diagnosis :**
 - a) There is no specific diagnostic test for ALS.
 - b) Clinically there should be a combination of UMN & LMN in **3 or more regions**.
 - c) An EMG (electromyogram) shows widespread fibrillation waves & exclude disorders of neuromuscular junction.
- **Treatment :**
 - No specific treatment, just supportive treatment.
 - **Riluzole** is a glutamate blocker : It may delay death by only 3 to 5 months.
- **Prognosis :** 80% mortality rate at 5 years, 100% at 10 years.
- **Causes of death :** Aspiration pneumonia & **respiratory failure**.
- **DD :** Myopathy, MG, Multiple Sclerosis, Poliomyelitis, syringomyelia, Hyperthyroidism.

Notice that the body is divided into 4 regions:

1. Bulbar : jaw, palate, larynx & tongue.
2. Cervical : neck, upper limbs & diaphragm.
3. Thoracic : back & abdomen
4. Lumbosacral : Lower limbs.

Stephen Hawking :

Physicist Stephen Hawking was born on January 8th 1942. In 1963, and shortly after his 21st birthday, Stephen Hawking **was diagnosed with ALS**. Doctors predicted he would only have two years to live. But, he has been living with ALS up till now and today he is 74 !! The disease began to break down neurological pathways but did not affect his mind. His intellect remained intact. In spite of being wheelchair bound



and dependent on a computerized voice system for communication, Stephen Hawking continues to combine family life. Hawking married twice and has three children. Stephen Hawking is regarded as one of the most brilliant theoretical physicists since Einstein. Professor Hawking has published many books tackling the fundamental questions about the universe and our existence e.g. **"A Brief History of Time"**.

He still inspires people with his will to not just survive, but to live and work. He has made us believe in, "anything and everything is possible, if you decide."

Quotes by Stephen Hawking :

- One of the basic rules of the universe is that nothing is perfect. Perfection simply doesn't exist. Without imperfection, neither you, nor I would exist.
- The greatest enemy of knowledge is not ignorance; it is the illusion of knowledge.
- Intelligence is the ability to adapt to change.
- My expectations were reduced to zero when I was 21. Everything since then has been a bonus.



- While there's life, there is hope.